POSTWAR LOAN FUND Now Available See page 425

ANNUAL MEETING, MAY 21, 22 and 23
HOTEL STATLER, BOSTON

1946 FUND CAMPAIGN
GIVE

The New England Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Volume 234 MARCH 21, 1946 Number 12

THE REPAIR OF CRANIAL DEFECTS WITH SPECIAL REFERENCE TO THE USE OF CANCELLOUS BONE 393
John T. B. Carmody

LACK OF A SPECIFIC URETHRAL LESION IN EXERTIONAL URINARY INCONTINENCE 400
S. Richard Muellner

PYOGENIC LIVER ABSCESS 403
J. Edward Flynn

MEDICAL PROGRESS: ABDOMINAL SURGERY 408
Arthur W. Allen

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL 416

EDITORIALS
New A.M.A. Program 424
"To do Something for the Welfare of Mankind" 424

DEPARTMENTS
Massachusetts Medical Society 425
Medicolegal Abstract 425
Notices 426

Owned and Published by the Massachusetts Medical Society

Published weekly at 8 Fenway, Boston 15, Massachusetts.
Domestic, $6 per year, 25c per copy (medical students, $3.50 per year); Canada, $7.04 (Boston funds); foreign, $8.52.
Entered as second-class matter, November 16, 1932, at the post office at Boston, Massachusetts, under the Act of March 3, 1879.
Copyright, 1946, by the Massachusetts Medical Society.

MASSACHUSETTS MEDICAL SOCIETY

OFFICERS AND STANDING COMMITTEES

President: REGINALD FITZ, Boston

Pres.-Elect: DWIGHT O'HARA, Waltham

Vice-Pres.: WILLIAM JASON MIXTER, Boston

Secretary: MICHAEL A. TIGHE, Lowell

Treas.: ELIOT HUBBARD, Jr., Cambridge

Asst.-Treas.: NORMAN A. WELCH, West Roxbury

Orator: FRANK H. LAHEY, Boston

Delegates to House of Delegates (A. M
DAVID D. SCANNELL, Jamaica
DWIGHT O'HARA, Waltham; CHARLE
MONGAN, Somerville; WALTER G. PHIP
Salem; CHARLES J. KICKHAM, Broo
LELAND S. McKITTRICK, Boston.

Executive Offices, 8 Fenway, Boston 15, Telephone, KEN 2094

Committee on Publications — *Chairman*, Richard M. Smith; James P. O'Hare, Conrad Wesselhoeft, Oliver Cope, John Fallon.

Committee on Arrangements — *Chairman*, Roy J. Heffernan; Sidney C. Wiggin, G. Guy Bailey, Harold G. Giddings, Robert L. Goodale.

Committee on Ethics and Discipline — *Chairman*, Ralph R. Stratton; William J. Brickley, Allen G. Rice, Fred R. Jouett, Archibald R. Gardner.

Committee on Medical Education — *Chairman*, Robert T. Monroe; George D. Henderson, Chester S. Keefer, Isaac R. Jankelson, Arthur W. Allen.

Committee on Membership — *Chairman*, Harlan F. Newton; Peirce H. Leavitt, Samuel N. Vose, Roy V. Baketel, William A. R. Chapin, William H. Allen,* H. Quimby Gallupe,* Albert E. Parkhurst.*

*Representing the supervising censors.

Committee on Public Health — *Chairman*, Roy J. Ward; Ernest Morris, Conrad Wesselhoeft, Elmer S. Bagnall, George L. Steele.

Committee on Medical Defense — *Chairman*, Arthur W. Allen; D. D. Gardner, William R. Morrison, Horatio Rogers, Ira M. Dixson.

Committee on Society Headquarters — *Chairman*, Frank R. Ober; G. G. Mixter, Michael A. Tighe, Daniel B. Reardon, Albert A. Hornor.

Committee on Finance — *Chairman*, Francis C. Hall; Ernest L. Charles F. Willinsky, Edward J. O'Brien, Jr., Peer P. Johnson.

Committee on Industrial Health — *Chairman*, Thomas L. Joseph C. Aub, Daniel L. Lynch, Louis R. Daniels, John G. Patrick E. Gear, Harold R. Kurth.

OFFICERS OF THE SECTIONS

Section of Medicine
Chairman, Albert A. Hornor, Boston; *vice-chairman*, Daniel J. Ellison, Lowell; *secretary*, Francis C. Hall, Boston.

Section of Surgery
Chairman, Charles F. Twomey, East Lynn; *secretary*, Alexander J. A. Campbell, Boston. **Executive Committee** — Stanley J. G. Nowak, Belmont and Boston (1 year); Edward L. Young, Jr., Brookline and Boston (2 years); E. Parker Hayden, Brookline and Boston (3 years).

Section of Pediatrics
Chairman, Floyd R. Smith, Pittsfield; *secretary*, Gerald N. Hoeffel, Cambridge. **Executive Committee** — *Chairman*, Philip H. Sylvester, Boston; James Marvin Baty, Belmont and Brookline; Leroy T. Stokes, Haverhill.

Section of Obstetrics and Gynecology
Chairman, Arthur F. G. Edgelow, Springfield; *vice-chairman*, W J. McDonald, Boston; *secretary*, George Van S. Smith, Brookline.

Section of Radiology
Chairman, George Levene, Chestnut Hill and Boston; *secretary*, Ed B. D. Neuhauser, Cambridge and Boston.

Section of Physiotherapy
Chairman, Arthur L. Watkins, Arlington and Boston; *secretary*, Ho Moore, Newton and Boston.

Section of Dermatology and Syphilology
Chairman, Bernard Appel, Lynn; *secretary*, Fenner A. Chace, Fall

Section of Anesthesiology
Chairman, Sidney C. Wiggin, Waban; *secretary*, Leo V. Hand, Highlands.

OFFICERS OF THE DISTRICT MEDICAL SOCIETIES FOR 1945–1946

Barnstable — *President*, Joseph N. Kelly, Orleans; *vice-presidents*, Harold F. Rowley, Harwich Port; *secretary*, Julius G. Kelley, Pocasset; *treasurer*, Frank Travers, Barnstable; *librarian*, Carroll H. Keene, Chatham; *executive councilor*, Paul M. Butterfield, Harwich; *legislative councilor*, Julius G. Kelley, Pocasset; *public-relations councilor*, William D. Kinney, Osterville.

Berkshire — *President*, Thomas H. Nelligan, Pittsfield; *vice-president*, N. Newall Copeland, Pittsfield; *secretary*, Daniel N. Beers, Pittsfield; *treasurer*, Theodore W. Jones, Pittsfield; *executive councilor*, Isaac S. F. Dodd, Pittsfield; *legislative councilor*, Clement F. Kernan, Pittsfield; *public-relations councilor*, Patrick J. Sullivan, Dalton.

Bristol North — *President*, William M. Stobbs, Attleboro; *vice-president*, Joseph V. Chatigny, Taunton; *secretary*, William J. Morse, Attleboro; *treasurer*, Charles E. Hoye, Taunton; *executive councilor*, William H. Allen, Mansfield; *legislative councilor*, Ralph M. Chambers, Taunton; *public-relations councilor*, James H. Brewster, Attleboro.

Bristol South — *President*, John C. Corrigan, Fall River; *vice-president*, S. Perry Wilde, New Bedford; *secretary and treasurer*, James E. Fell, Fall River; *executive councilor*, Edwin D. Gardner, New Bedford.

Essex North — *President*, Percy J. Look, Andover; *vice-president*, Guy L. Richardson, Haverhill; *secretary*, Harold R. Kurth, Methuen; *treasurer*, Lawrence Murphy, Newburyport; *executive councilor*, Frank W. Snow, Newburyport; *legislative councilor*, Edward H. Ganley, Methuen; *public-relations councilor*, Harold R. Kurth, Methuen.

Essex South — *President*, William R. Irving, Gloucester; *vice-president*, John W. Trask, Lynn; *secretary*, Henry D. Stebbins, Marblehead; *treasurer*, Charles F. Deering, Danvers; *executive councilor*, Walter G. Phippen, Salem; *legislative councilor*, Charles A. Worthen, Lynn; *public-relations councilor*, Loring Grimes, Lynn.

Franklin — *President*, Kenneth H. Rice, South Deerfield; *vice-president*, John B. Temple, Shelburne Falls; *secretary and treasurer*, Harry L. Craft, Ashfield; *executive councilor*, William J. Pelletier, Turners Falls; *legislative councilor*, Howard M. Kemp, Greenfield; *public-relations councilor*, John E. Moran, Greenfield.

Hampden — *President*, Merrill F. Hosmer, Springfield; *vice-president*, Patrick E. Gear, Holyoke; *secretary and treasurer*, George C. Steele, West Springfield; *executive councilor*, William A. R. Chapin, Springfield; *legislative councilor*, Arthur Riordan, Indian Orchard; *public-relations councilor*, Patrick E. Gear, Holyoke.

Hampshire — *President*, Arthur N. Ball, Northampton; *vice-president*, Elmer H. Copeland, Northampton; *secretary and treasurer*, Mary P. Snook, Worthington; *librarian*, Abbie M. O'Keefe, Northampton; *executive councilor*, Joseph D. Collins, Northampton; *legislative councilor*, Arthur N. Ball, Northampton; *public-relations councilor*, William M. Dobson, Northampton.

Middlesex East — *President*, Justin L. Anderson, Reading; *vice-president*, Walter H. Flanders, Melrose; *secretary*, Roy W. Layton, Melrose; Albert E. Small, Melrose; *executive councilor*, Edward M. Halligan, *legislative councilor*, John M. Wilcox, Woburn; *public-relations* Milton J. Quinn, Winchester.

Middlesex North — *President*, Adam E. Shaw, Lowell; *vice-pres* William F. Ryan, Lowell; *secretary*, Brendan D. Leahey, Lowell; Mason D. Bryant, Lowell; *executive councilor*, William F. Ryan, *legislative councilor*, Archibald R. Gardner, Lowell; *public-relations* Daniel J. Ellison, Lowell.

Middlesex South — *President*, Harold G. Giddings, Newton C *vice-presidents*, John F. Casey, Allston; *secretary*, Alexander A. Levi, Centre; *treasurer*, Fabyan Packard, Belmont; *orator*, Robert E. Gross, ham; *executive councilor*, Harold G. Giddings, Newton Centre; *councilor*, Edward J. O'Brien, Jr., Brighton; *public-relations councilor*, M. Morrison, Waban.

Norfolk — *President*, Dean S. Luce, Canton; *vice-president*, Cauley, Dorchester; *secretary*, Basil E. Barton, West Roxbury; Frederick Reis, Jamaica Plain; *executive councilor*, Charles J. Kickham, line; *legislative councilor*, Humphrey L. McCarthy, Brookline; *public-councilor*, Norman A. Welch, West Roxbury.

Norfolk South — *President*, Frank W. Crawford, Holbrook; *president*, David L. Belding, Hingham; *secretary*, Ebenezer K. Jenkins, tree; *treasurer*, Francis G. King, North Quincy; *executive councilor*, Reardon, Quincy; *legislative councilor*, David L. Belding, Hingham; *relations councilor*, Nahum R. Pillsbury, Braintree.

Plymouth — *President*, Paul B. Kelly, Plymouth; *vice-president*, H. Pulsifer, Whitman; *secretary*, Ralph C. McLeod, Brockton; Rudolph A. Kruger, Brockton; *executive councilor*, Peirce H. Leavitt, ton; *legislative councilor*, John J. McNamara, Brockton; *public-councilor*, Charles D. McCann, Brockton.

Suffolk — *President*, Donald Munro, Boston; *vice-president*, C. Lund, Boston; *secretary*, Robert L. Goodale, Boston; *treasurer*, Richa Eustis, Boston; *executive councilor*, Alexander J. A. Campbell, Boston; *lative councilor*, William E. Browne, Boston; *public-relations councilor*, A. Hornor, Boston.

Worcester — *President*, Charles R. Abbott, Clinton; *vice-pres* Lester M. Felton, Worcester; *secretary*, Leslie P. Leland, Worcester; Edward P. Disbrow, Worcester; *executive councilor*, Bancroft C. Wh Worcester; *legislative councilor*, Lester M. Felton, Worcester; *public-* councilor, John Fallon, Worcester.

Worcester North — *President*, Rudolph F. Bachmann, Fitchb *vice-presidents*, Francis A. Reynolds, Athol; *secretary*, James G. Sim Fitchburg; *treasurer*, Frederick H. Thompson, Jr., Fitchburg; *executive* cilor, C. Bertram Gay, Fitchburg; *legislative councilor*, Donald B. Ches Athol; *public-relations councilor*, James V. McHugh, Leominster.

The New England
Journal of Medicine

Copyright, 1946, by the Massachusetts Medical Society

Volume 234 MARCH 21, 1946 Number 12

THE REPAIR OF CRANIAL DEFECTS WITH SPECIAL REFERENCE TO THE USE OF CANCELLOUS BONE*

COMMANDER JOHN T. B. CARMODY (MC), U.S.N.R.†

THE treatment of defects of the cranial bones is a problem as old as cranial surgery. All types of material have been used, and the search for a completely satisfactory substance continues. The war has stimulated such endeavor, and there is no doubt that vast strides have been made. There has been a strong tendency toward the use of metal. Almost all metals have been tried in one form or another, each having its advocates and showing advantages over the others, but each possessing distinct disadvantages. Formerly, the most successful and most popular materials were silver and vitallium,[1] but other substances such as celluloid have had their followers. The repair of large cranial defects by silver plates has been successful for varying periods of time, but the majority of such plates have had to be replaced. Generally speaking, the use of silver is a thing of the past. In 1937, it was noted that vitallium caused only negligible tissue reaction and gave evidence of great promise,[1–4] but it was difficult to work with, and although still used this material has been superseded by tantalum.[5–7]

Burch, in 1938, first saw the surgical possibilities of tantalum,[7] and Pudenz,[8] in 1942, emphasized its future in neurosurgery in a paper on tissue tolerance of the metal in cats. It was also shown that a thin, filmy, translucent, gelatinous membrane enveloped the metal within forty-eight hours, thus tending to prevent fixation to the nervous tissue.[9] On the other hand, some question has arisen through the experiments of Delarue,[10] who, working with dogs, proved that tantalum could become attached to the cerebral cortex by firm adhesions and that the adjacent arachnoid and dura could become thickened. Recently, Robertson and Peacher[11] have found no such indication of tissue reaction in human beings, but there is evidence that such a complication may occur, as indicated in one of the cases reported in this paper.

Nevertheless, tantalum has been and continues to be the most satisfactory metal used in cranioplasty. It is light, nonmagnetic and chemically inert, is easily molded and does not require preliminary casting. It is soft enough so that drill holes can easily be placed at the time of operation, and no special instruments are necessary to handle it. In summary, tantalum offers inertness, mobility and strength — three factors not present in the metals previously used.

In the matter of basic requirements for an alloplastic material, it appears that the goal has been reached. Recently, several impressive series of cases receiving cranioplasty have appeared in the literature.[9, 12, 13] The immediate results in many cases have been remarkable, but in spite of initial perfection, some evidence is accumulating that tantalum is not suitable in all cases. Heavily scarred areas that have remained intact for varying periods have gradually broken down, and once this process has begun, the basic inertness of the metal does not prevent it from being extruded like any foreign body. Again, the possibility of cortical fixation has not been completely disproved. Other disadvantages are gradually taking shape, such as the preclusion by a plate of any future x-ray studies. It is true that some of these disadvantages may arise from the technical difficulties of cranioplasty rather than from the metal itself. Also, it cannot be denied that the result of enthusiasm for cranioplasty has been that many cranial defects have been subjected to so-called plating that in former years and under different circumstances would have been let alone. Significantly, in this group are found cases with more complaints following repair than before it. Much has been written concerning the indication for cranioplasty, and almost any case of skull defect can be made to meet such requirements.[6, 12, 13]

It seems more to the point to emphasize the contraindications. The clinical cases exhibiting them fall into two main categories — those with a simple "hole in the head" associated with no complaints, and those in which the complaints are consistent with functional involvement. There

*This article has been released for publication by the Division of Publications of the Bureau of Medicine and Surgery of the United States Navy. The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

†Neurosurgeon, Worcester City and Worcester Memorial hospitals, and assistant in neurosurgery, Massachusetts General Hospital (on leave of absence).

are, of course, other minor groups of cases, such as those with sizable retained foreign bodies, in which

FIGURE 1. *Case 1.*

This shows the condition of the patient's wound on admission. The presenting portions of the perforated plate are readily seen. It illustrates the manner in which the heavily scarred, thinned-out areas break down. Three days later the rest of the scar gave way.

the advisability of cranioplasty is questionable. Opinion concerning its value when epilepsy is present is divided. At one extreme, Gardner[12] believes that cranioplasty has been most beneficial. The time interval in the series of cases subsequently reported has not been sufficient to warrant any definite conclusion, and although some suggestive improvement has occurred, it must be remembered that a cranial procedure of any type frequently effects a temporary change in the convulsive state.

The following 2 cases are summarized to illustrate some of the inadequacies of tantalum. They are presented not from the point of view of criticism of treatment but in an attempt to show that the use of bone in cranioplasty is not obsolete and that in certain cases this material should in fact be the one of choice.

CASE REPORTS

CASE 1. A 29-year-old veteran, was admitted to the hospital on February 16, 1945, with a diagnosis of meningitis. He complained of severe headache, a draining head wound and a stiff neck. He had been wounded in the head by a bomb fragment on September 23, 1943, and had developed successively a herniation of the right frontal cerebral hemisphere, right frontal osteomyelitis and epilepsy. Subsequently, several operations had been performed, including as a final procedure the insertion of a large tantalum plate. The patient was discharged from the service on January 6, 1945, at which time his general condition was excellent. The head wound had completely healed. During the next month, he experienced slight headaches and a feeling of tightness involving the right frontal region. These symptoms increased in severity and culminated in the breaking down of a portion of the heavily scarred scalp.

Physical examination on admission showed an acutely ill patient. The temperature was 101°F., and the pulse 11[illegible]. The neck was slightly rigid, and a profuse thin, purulent discharge was issuing from the several presenting perforations in the exposed areas of the tantalum plate (Fig. 1). The remainder of the examination was negative with the exception of a left-sided hyper-reflexia. Cultures were positive for a hemolytic staphylococcus.

The infection was readily controlled by chemotherapy but further breakdown of the scar continued. X-ray examination showed a large and exceptionally well molded

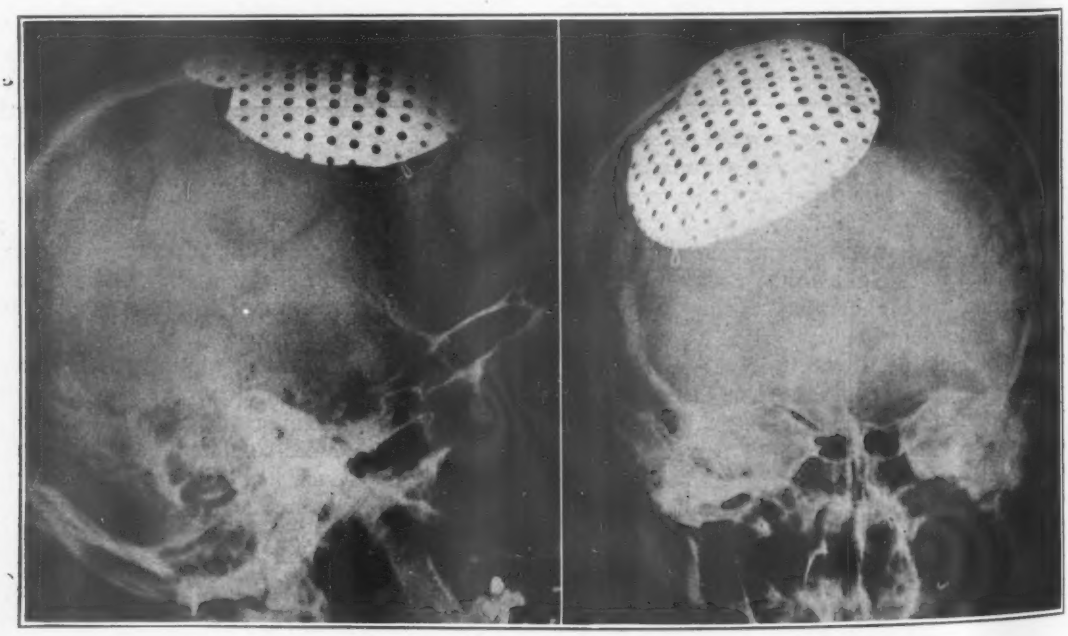

FIGURE 2. *Case 1.*

These films show the size of the tantalum plate. Many old fracture lines remain visible, indicating the extensiveness of the original injury.

and fitted tantalum plate (Fig. 2). It was decided to save as much of the plate as possible. It seemed to be a foregone conclusion that a subsequent total cranioplasty would be

doomed from the beginning. Consequently, a section of the plate underlying the main scar areas was removed. This allowed the soft-tissue edges to overhang the plate, but in spite of tissue contact, further recession with attempted extrusion of the plate ensued, and as a result it was surgically removed. At the time it was noted that a multiloculated space, some areas being fully 2 cm. in depth, lay between the plate and the intracranial structures and encompassed the entire undersurface of the plate. This space was partitioned by thin tissue bands, with the exception of the frontal periphery, which was adherent to the dura. Whether this picture was entirely the result of the infection could not be determined. The scalp was firmly attached to the plate in a different manner than has been noted with nonperforated plates.

FIGURE 3. *Case 1.*

This film is a right lateral view that was taken three months postoperatively. The graft is solid, and the inner bone surface, as seen in the stereoscopic films, forms a smooth and even contour.

The only form of treatment for the next several weeks was frequent irrigations with penicillin. The multiloculated space gradually filled in. X-ray examination showed no evidence of bone infection.

A bone graft of cancellous chips from the ilium was de-

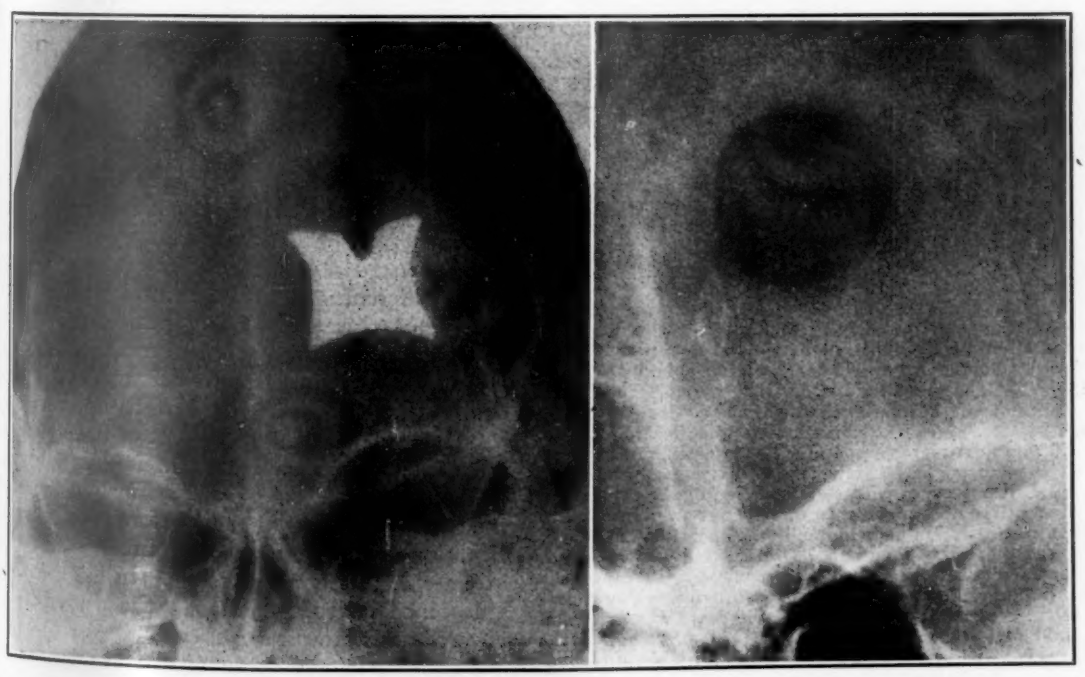

FIGURE 4. *Case 2.*

A is a film showing the tantalum plate in position over the skull defect; the left frontal sinus is less distinct than the right one. B is a film taken two months after operation; the graft was firm and solid and had effected the proper contour to the forehead.

cided on and was carried out. Prompt healing followed, and the last x-ray film showed that the graft was intact and united (Fig. 3). At the present time the patient has no complaints referable to the head. Clinically the grafted area is sound. There is no pulsation, and the patient no longer feels as though, in his words, something were in his head that did not belong there.

CASE 2. A 20-year-old seaman was admitted to the hospital on May 8, 1945, with the complaints of headache and of swelling and tenderness in the left frontal region. He had had a frontal sinusitis on August 29, 1944, and had subsequently developed a left frontal brain abscess. The abscess was drained on November 5, and a small tantalum plate

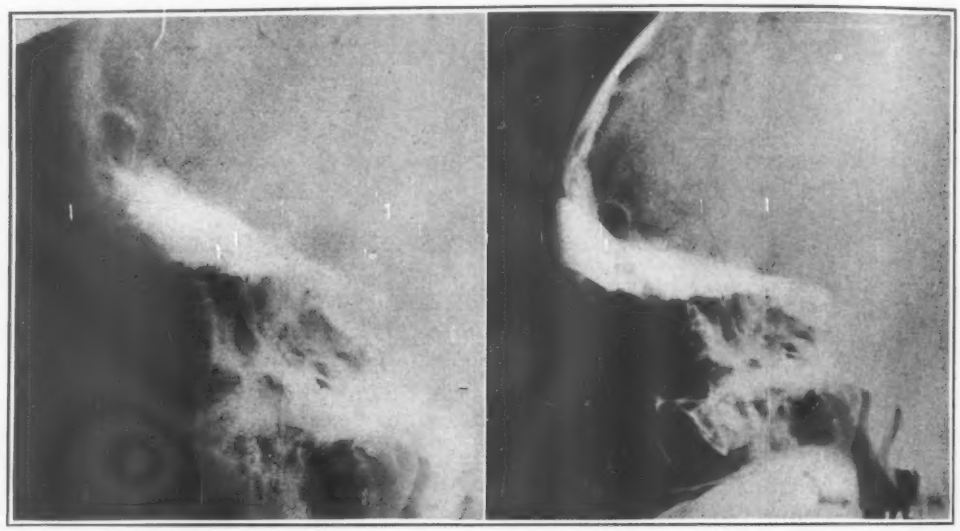

FIGURE 5. *Case 3.*

A shows a defect of the left orbital ridge and frontal sinus, with an unsatisfactory attempt at repair with bone; the bone graft was loose, sagged into the soft tissues of the orbit, with marked deformity, and caused severe pain due to tension on the supraorbital nerve. B *is a film taken after removal of the graft and repair of the defect by a contour-fitting tantalum plate.*

was inserted to prevent deformity. The patient showed prompt and immediate improvement and was discharged to full duty on April 26, 1945.

On admission he stated that he had been well except when he "caught cold." With the onset of each upper respiratory infection, he noted severe headache, swelling and tenderness about the site of the operation. These attacks at first subsided within a few days, but the last one was severe and resulted in admission to the sick list.

Physical examination showed an acutely ill young man. There was moderate swelling of the entire left frontal area. The tantalum plate could be palpated, and there was marked tenderness about its edges. There were no abnormal neurologic signs. The spinal fluid was within normal range with the exception of the pressure, which was equivalent to 250 mm. of water. X-ray examination showed the presence of a small tantalum plate (Fig. 4*A*). The lateral film suggested that the plate might be riding, and this was confirmed by the onset of pulsations with the regression of the swelling.

Conservative treatment was carried out until the acute phase of the infection had subsided. At operation on June 6, 1945, the plate was found to be riding and was removed. At the same time the defect was repaired with cancellous bone from the ilium. On the 6th postoperative day pulsations had ceased. Within 2 weeks the graft was firm, and the final film, taken 2 months later (Fig. 4*B*) showed the bone graft to be intact and united firmly with the surrounding bone. The patient was followed for 2 months and remained free of complaints.

The operative technic in this procedure does not differ from that of any other method so far as the preparation of the site of the bone defect is concerned. There are, however, several points that must be observed if rapid healing is to occur. In the first place, all scarred areas should be carefully avoided in making the incision, unless they are of such size and location that they can be resected at the time. In many cases the fractures have been extensively compounded, with severe loss of soft tissue, resulting in heavy scar formation and fixation to the underlying tissues, including the dura or the cortex. When such areas, or even minor portions of them, have been included in the incision, some degree of incisional breakdown invariably follows. In many cases it is not feasible to attempt plastic correction of scarring as a preliminary step to defect repair, because of the extensive and complete adherence of the intracranial tissues to the overlying structure. Many patients have undergone several operative procedures, and the advisability of any further preliminary plastic repair before that of the defect has been accomplished is questionable. The treatment of these scars may be a major problem and is in itself a subject requiring separate attention.[13] Finally, the wound should be completely healed before grafting is done. Occasionally, in exceptionally involved defects, as in Case 1, this is not possible. In this event, convalescence is lengthened and the danger of bone absorption is increased.

Following the making of the flap incision, the aponeurosis is dissected from the periosteum to the edge of the bone defect, and the scar, which may begin at the margin of the defect, is carefully dissected from the dura. It is most important

not to pierce through the scarred area to its external surface. This is an extremely easy thing to do, and is almost certain to result in tissue breakdown postoperatively. When the lower margin of the defect has been exposed and completely freed, all bleeding points — and there are often many, depending on the size and character of the scar — may be controlled more completely and easily with fibrin foam-packs than by any other combination of methods. Freshening up the bony edge around the defect should be avoided. The danger of diploic bleeding outweighs any suspected advantage that such a procedure may have.

With the bed prepared and all bleeding controlled, cancellous chips approximately 1 cm. in diameter are placed in mosaic layers, entirely filling the defect. Chips of this size are readily obtained by biting them off from the graft with a rongeur. As a final step, when possible, the periosteum surrounding the exposed bone defect is incised and turned back over the borders partially enclosing the cancellous chips. Mowlem[14] believes that this is not necessary and prefers to have the chips in direct contact with the denuded bone. The scalp flap is sutured and a light compression dressing is applied.

Postoperatively, formation of a hematoma occasionally occurs in spite of the utmost precautions before closure. Local application of heat, accompanied by drainage at the most dependent region of the incision, results in quick resolution. When an extensive loss of underlying soft tissue has occurred, the original scar may break down. Irrigation with penicillin in a 1:1000 concentration is followed by rapid healing. This result is a decided contrast to the problem that arises when metal of any kind is used as the underlying material.

So far as the graft is concerned, firm union is present clinically in two or three weeks. Generally the x-ray picture in the early stage is that of intact and living bone without confirmatory evidence of union. The proper contour of the graft is readily maintained by the pressure of the overlying scalp against the easily molded mass of cancellous chips. Contour adaptability of the graft is the greatest single advantage of this method. The fact that Mowlem[14] has had marked success in extensive facial-bone defects emphasizes the ease with which cancellous bone can be molded to fit the occasion.

That absorption of some elements of the graft may take place cannot be denied. This has occurred in some of this series of cases, but in none to the extent that the graft has lost its primary point of obliterating the defect. Actually, when absorption did take place only the chips that could be considered excessive were singled out, and in such a manner that the graft gradually progressed in contour and thickness to that of the normal adjacent bone.

The method of taking the graft is simple and can be carried out in a few minutes. The crest of the ilium is exposed and the flat surface of the bone is laid bare. The outer cortical layer is split with a rotary bone saw and turned down. Thin strips of the medulla are sliced free, and when a sufficient amount has been obtained the cortical layer is replaced and sutured into its former position. The hip wounds heal quickly, and there have been no complications or complaints other than soreness for the first two or three days.

Discussion

It is not the purpose of this paper to condemn or undermine the use of tantalum in cranioplasty, but rather to advance the cause of cancellous-bone grafting in selected cases. An attempt has been made to show that cancellous bone may be the material of choice under certain circumstances and that its use should not be relegated to the past.

It is difficult to conceive of cancellous bone's fulfilling satisfactorily the obligation of repair either in exceptionally large defects or in those whose location precludes its use. With respect to the first type of case, the use of cancellous bone must be self-limiting. Sufficient bone to repair small and medium-sized defects is readily obtained from the ilium, as outlined above, whereas to take the amount of bone required for the larger defects would transform a minor procedure into one of major proportions. In defects of the orbital ridge and frontal sinuses, tantalum seems preferable, although Mowlem[14] has used cancellous bone in this location with excellent results. Figures 5 and 6 depict a problem of this type.

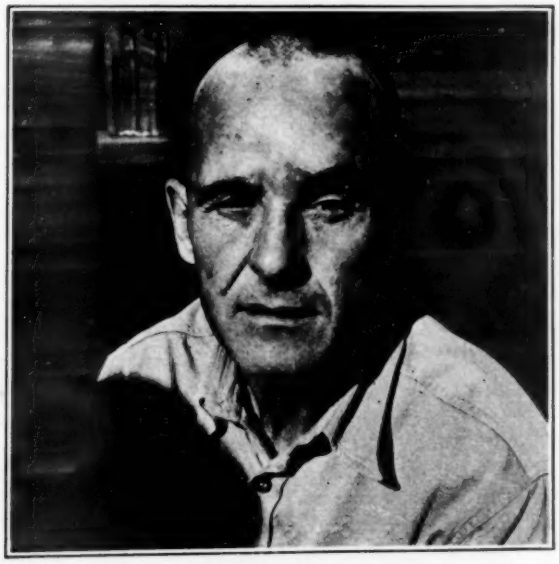

Figure 6. *Case 3.*
This is a photograph of the patient taken two weeks postoperatively. The defect in the left orbital ridge and frontal sinus has been corrected.

On the other hand, in both simple and complicated medium-sized defects of the vault, cancellous-bone grafting is in every particular the best method. The last 7 cases receiving cranioplasty in this hospital have been treated in this manner, and with one exception healing has been prompt. In the excepted case, there was a minor breakdown of tissue involving a portion of the original scar. Irrigations with penicillin were used, and rapid healing occurred. In all cases the grafts were clinically solid within two or three weeks. Defect pulsation disappeared in approximately one week, with a coincidental subsidence of the defect caused by the complaints. In defect areas in which deformity had been marked, proper and satisfactory contour healing occurred; Figures 7 and 8 illustrate such a case. Furthermore, some patients with plates have said that they are constantly aware of the presence of a foreign body — in their own words, "something that does not belong there." It seems logical to assume that no matter how inert a material may be it is still a foreign body, and if its presence can be avoided by the use of autogenous material, this should be employed. Another factor of importance

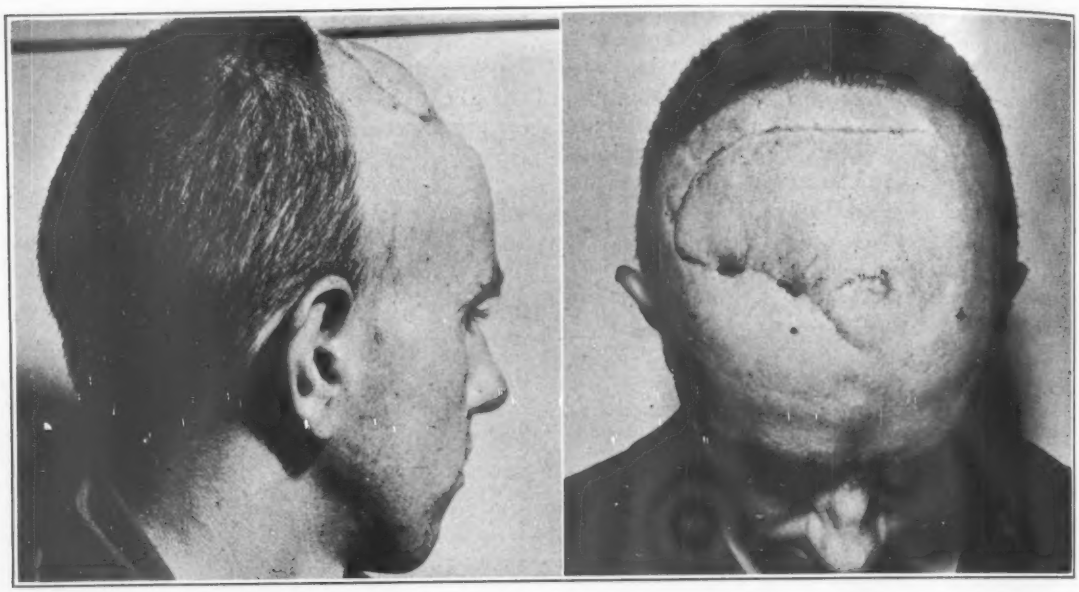

FIGURE 7. *Case 4.*
These photographs show a medium-sized defect, with contour deformity, in the right frontal bone.

FIGURE 8. *Case 4.*
These photographs were taken two weeks following a cranioplasty with cancellous bone. The contour deformity had been corrected, pulsations had ceased, and the graft was solid.

is that of all methods of repair cancellous-bone grafting is certainly the simplest and easiest. Plating by mortising the periphery of the bone defect may be an undertaking of considerable magnitude.[15, 16] Fixation or wedging is simple when the exposure is easily achieved, but with involvement of the squamous portion of the temporal bone, even these methods become complicated. When impressions of the defect are made for casting, a two-stage operation is the rule. Simple fixation by tantalum screws reduces the operation to the level of grafting, but here again such a method is not always applicable.

Attention must be called to the fact that other materials than metals are being used in cranioplasty. Methyl acrylic, with which we have had no experience, is receiving support by its advocates,[13] but its use also requires a longer procedure and a more elaborate setup. That it has any outstanding advantages over tantalum, with the exception of its being radiolucent, seems doubtful.

Recently, Mowlem[14] has published a series of 75 cases of bone grafts in which cancellous chips were used exclusively. In most of the cases dealt with there were facial and long-bone defects, but there was 1 case of frontal-bone defect in which complete clinical healing with firm union occurred in ten days. This case has been followed for three years, during which period the graft has remained solid, with no evidence of absorption.

Mowlem attributes the marked success of cancellous bone for grafting to its highly vascular character. He believes further that fragmentation is a definite aid in the survival of these grafts, on the theory that a greater proportion of the bone cells becomes accessible to early vascularization.

The chief objections in the past to the use of bone in defect repairs — namely, the extensiveness of the operation, often necessitating two stages, and the extreme difficulty of obtaining the proper contour effect — have been overcome by the use of cancellous bone. Furthermore, if the size of the cranial defect is kept at a minimum at the time of the initial débridement, as has been frequently advocated, many unnecessarily large defects requiring the use of alloplastic materials can be avoided.[17, 18] This in itself as a preventive measure would do much to reduce the number of cases requiring extensive cranioplastic measures.

Conclusions

Autogenous cancellous bone is the material of choice in small and medium-sized defects of the skull requiring cranioplasty.

The repair of cranial defects with tantalum is at present the procedure of choice when the defects are of such size that a sufficient amount of bone cannot be obtained without an extensive secondary procedure, or when the site of the defect renders tantalum the more suitable material.

References

1. Venable, C. S., Stuck, W. G., and Beach, A. Effects on bone of presence of metals: based upon electrolysis: experimental study. *Ann. Surg.* **105**:917-938, 1937.
2. Beck, C. S. Repair of defects of skull by ready made vitallium plates. *J. A. M. A.* **118**:798, 1942.
3. Geib, F. W. Vitallium skull plates. *J. A. M. A.* **117**:8-12, 1941.
4. Kazanjian, V. H., and Holmes, E. M. Reconstruction after radical operation for osteomyelitis of frontal bone: experience in 38 cases. *Surg., Gynec. & Obst.* **79**:397-411, 1944.
5. Burke, G. L. Corrosion of metals in tissues: and introduction to tantalum. *Canad. M. A. J.* **43**:125-128, 1940.
6. Fulcher, O. H. Tantalum as metallic implant to repair cranial defects: preliminary report. *J. A. M. A.* **121**:931-933, 1943.
7. Burch. Cited by Pudenz, R. H. Repair of cranial defects with tantalum: experimental study. *J. A. M. A.* **121**:478-481, 1943.
8. Pudenz, R. H., and Odom, G. L. Meningocerebral adhesions: experimental study of effect on human amniotic membrane, amnioplastin, beef allantoic membrane, cargile membrane, tantalum foil and polyvinyl alcohol films. *Surgery* **12**:318-344, 1942.
9. Robertson, R. C. L. Repair of cranial defects with tantalum. *J. Neurosurg.* **1**:227-236, 1944.
10. Delarue, N. C., Linell, E. A., and McKenzie, K. G. Experimental study on use of tantalum in subdural space. *J. Neurosurg.* **1**:239-242, 1944.
11. Robertson, R. C. L., and Peacher, W. G. Use of tantalum foil in subdural space. *J. Neurosurg.* **7**:218-284, 1945.
12. Gardner, W. J. Closure of defects of skull with tantalum. *Surg., Gynec., & Obst.* **80**:303-312, 1945.
13. Woodhall, B., and Spurling, R. G. Tantalum cranioplasty for war wounds of skull. *Ann. Surg.* **121**:649-671, 1945.
14. Mowlem, R. Cancellous chip bone-grafts: report on 75 cases. *Lancet* **2**:746, 1944.
15. Gurdjian, E. S., Webster, J. E., and Brown, J. C. Impression technique for reconstruction of large skull defects. *Surgery* **14**:876-881, 1943.
16. Hemberger, A. J., Whitcomb, B. B., and Woodhall, B. Technique of tantalum plating of skull defects. *J. Neurosurg.* **2**:21-25, 1945.
17. Carmody, J. T. B. Management of major compound fractures of skull vault. *Am. J. Surg.* **57**:389-405, 1942.
18. *Idem.* War wounds of head. *U. S. Nav. M. Bull.* **41**:1227-1239, 1943.

On the other hand, in both simple and complicated medium-sized defects of the vault, cancellous-bone grafting is in every particular the best method. The last 7 cases receiving cranioplasty in this hospital have been treated in this manner, and with one exception healing has been prompt. In the excepted case, there was a minor breakdown of tissue involving a portion of the original scar. Irrigations with penicillin were used, and rapid healing occurred. In all cases the grafts were clinically solid within two or three weeks. Defect pulsation disappeared in approximately one week, with a coincidental subsidence of the defect caused by the complaints. In defect areas in which deformity had been marked, proper and satisfactory contour healing occurred; Figures 7 and 8 illustrate such

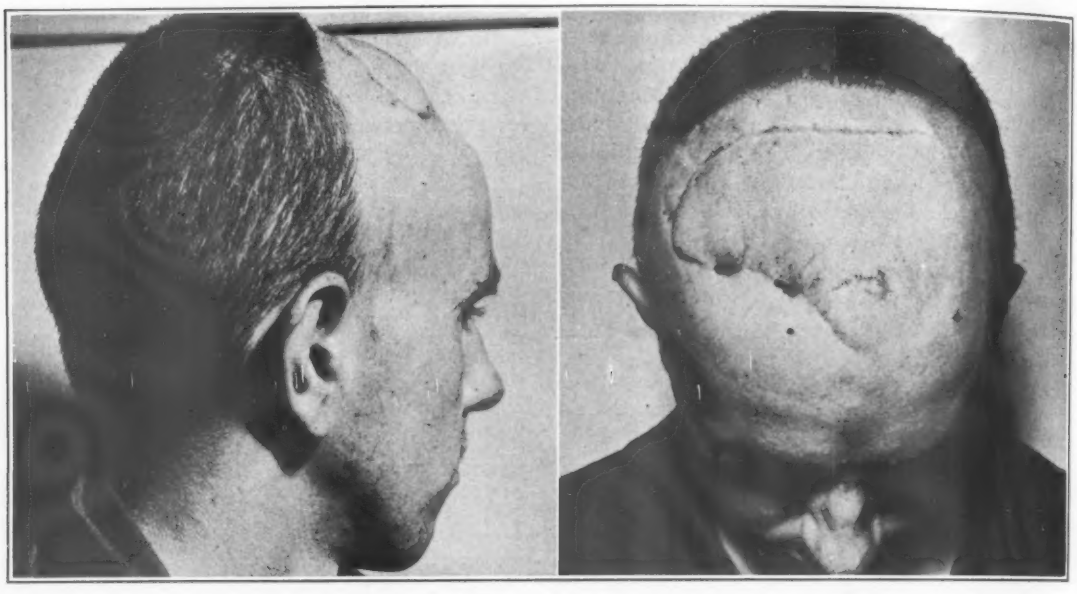

FIGURE 7. *Case 4.*
These photographs show a medium-sized defect, with contour deformity, in the right frontal bone.

case. Furthermore, some patients with plates have said that they are constantly aware of the presence of a foreign body — in their own words, "something that does not belong there." It seems logical to assume that no matter how inert a material may be it is still a foreign body, and if its presence can be avoided by the use of autogenous material, this should be employed. Another factor of importance

FIGURE 8. *Case 4.*
These photographs were taken two weeks following a cranioplasty with cancellous bone. The contour deformity had been corrected, pulsations had ceased, and the graft was solid.

is that of all methods of repair cancellous-bone grafting is certainly the simplest and easiest. Plating by mortising the periphery of the bone defect may be an undertaking of considerable magnitude.[15, 16] Fixation or wedging is simple when the exposure is easily achieved, but with involvement of the squamous portion of the temporal bone, even these methods become complicated. When impressions of the defect are made for casting, a two-stage operation is the rule. Simple fixation by tantalum screws reduces the operation to the level of grafting, but here again such a method is not always applicable.

Attention must be called to the fact that other materials than metals are being used in cranioplasty. Methyl acrylic, with which we have had no experience, is receiving support by its advocates,[13] but its use also requires a longer procedure and a more elaborate setup. That it has any outstanding advantages over tantalum, with the exception of its being radiolucent, seems doubtful.

Recently, Mowlem[14] has published a series of 75 cases of bone grafts in which cancellous chips were used exclusively. In most of the cases dealt with there were facial and long-bone defects, but there was 1 case of frontal-bone defect in which complete clinical healing with firm union occurred in ten days. This case has been followed for three years, during which period the graft has remained solid, with no evidence of absorption.

Mowlem attributes the marked success of cancellous bone for grafting to its highly vascular character. He believes further that fragmentation is a definite aid in the survival of these grafts, on the theory that a greater proportion of the bone cells becomes accessible to early vascularization.

The chief objections in the past to the use of bone in defect repairs — namely, the extensiveness of the operation, often necessitating two stages, and the extreme difficulty of obtaining the proper contour effect — have been overcome by the use of cancellous bone. Furthermore, if the size of the cranial defect is kept at a minimum at the time of the initial débridement, as has been frequently advocated, many unnecessarily large defects requiring the use of alloplastic materials can be avoided.[17, 18] This in itself as a preventive measure would do much to reduce the number of cases requiring extensive cranioplastic measures.

Conclusions

Autogenous cancellous bone is the material of choice in small and medium-sized defects of the skull requiring cranioplasty.

The repair of cranial defects with tantalum is at present the procedure of choice when the defects are of such size that a sufficient amount of bone cannot be obtained without an extensive secondary procedure, or when the site of the defect renders tantalum the more suitable material.

References

1. Venable, C. S., Stuck, W. G., and Beach, A. Effects on bone of presence of metals: based upon electrolysis: experimental study. *Ann. Surg.* **105**:917-938, 1937.
2. Beck, C. S. Repair of defects of skull by ready made vitallium plates. *J. A. M. A.* **118**:798, 1942.
3. Geib, F. W. Vitallium skull plates. *J. A. M. A.* **117**:8-12, 1941.
4. Kazanjian, V. H., and Holmes, E. M. Reconstruction after radical operation for osteomyelitis of frontal bone: experience in 38 cases. *Surg., Gynec. & Obst.* **79**:397-411, 1944.
5. Burke, G. L. Corrosion of metals in tissues: and introduction to tantalum. *Canad. M. A. J.* **43**:125-128, 1940.
6. Fulcher, O. H. Tantalum as metallic implant to repair cranial defects: preliminary report. *J. A. M. A.* **121**:931-933, 1943.
7. Burch. Cited by Pudenz, R. H. Repair of cranial defects with tantalum: experimental study. *J. A. M. A.* **121**:478-481, 1943.
8. Pudenz, R. H., and Odom, G. L. Meningocerebral adhesions: experimental study of effect on human amniotic membrane, amnioplastin, beef allantoic membrane, cargile membrane, tantalum foil and polyvinyl alcohol films. *Surgery* **12**:318-344, 1942.
9. Robertson, R. C. L. Repair of cranial defects with tantalum. *J. Neurosurg.* **1**:227-236, 1944.
10. Delarue, N. C., Linell, E. A., and McKenzie, K. G. Experimental study on use of tantalum in subdural space. *J. Neurosurg.* **1**:239-242, 1944.
11. Robertson, R. C. L., and Peacher, W. G. Use of tantalum foil in subdural space. *J. Neurosurg.* **7**:218-284, 1945.
12. Gardner, W. J. Closure of defects of skull with tantalum. *Surg., Gynec., & Obst.* **80**:303-312, 1945.
13. Woodhall, B., and Spurling, R. G. Tantalum cranioplasty for war wounds of skull. *Ann. Surg.* **121**:649-671, 1945.
14. Mowlem, R. Cancellous chip bone-grafts: report on 75 cases. *Lancet* **2**:746, 1944.
15. Gurdjian, E. S., Webster, J. E., and Brown, J. C. Impression technique for reconstruction of large skull defects. *Surgery* **14**:876-881, 1943.
16. Hemberger, A. J., Whitcomb, B. B., and Woodhall, B. Technique of tantalum plating of skull defects. *J. Neurosurg.* **2**:21-25, 1945.
17. Carmody, J. T. B. Management of major compound fractures of skull vault. *Am. J. Surg.* **57**:389-405, 1942.
18. *Idem.* War wounds of head. *U. S. Nav. M. Bull.* **41**:1227-1239, 1943.

LACK OF A SPECIFIC URETHRAL LESION IN EXERTIONAL URINARY INCONTINENCE*

S. Richard Muellner, M.D.†

BOSTON

IT IS generally believed that exertional urinary incontinence is due to trauma of childbirth. Although there is no agreement on the precise structure that has been injured, prevailing opinion ascribes this symptom to tears or fixations of the urethral sphincters or to attenuations of the bladder and urethral supports. The signs of obstetric trauma, however, are often not discernible on pelvic examination or at operation, and even when present they frequently cannot be correlated with the degree of urinary incontinence. Thus, marked urinary leakage may occur in patients with intact pelvic structures, whereas women who on exertion lose only a drop or two of urine may have a urethra and a bladder base that have obviously lost their normal attachments. Furthermore, a history of obstetric trauma may be absent, and it is well known that some women who have never borne children suffer from exertional incontinence. These facts suggest that the present ideas concerning the etiology of this condition require revision, and this need furnished the incentive for the present investigation.

A study of 140 women forms the basis of this report. Ninety-five patients had exertional incontinence, and 45 continent women who had had obstetric sequelae, such as cystocele, laceration of the perineum and prolapse of the uterus, were selected for control subjects. It will be seen from Table 1 that the age, the obstetric experience and the bladder capacity of both groups were closely similar. The use of such a large control group was considered essential for the proper evaluation of the findings ordinarily associated with exertional incontinence.

Table 1. *Comparative Data in Patients with Incontinence and in Control Patients.*

Datum	Patients with Incontinence	Control Patients
Age (in years):		
Average	44	54
Youngest	24	21
Oldest	68	72
Parity:		
Average	5	5
Extent	1–12	1–10
Bladder capacity (in cubic centimeters):		
Average	550	592
Largest	1150	950
Smallest	300	250

The data obtained consisted of a detailed history, a complete physical examination, cystometry, cystourethroscopy, cystography and urethrography. At the cystoscopic examination, particular attention was paid to the performance of the internal sphincter when closing and opening. In a few cases fluoroscopy of the bladder filled with a contrast medium (Diodrast) was done to study detrusor behavior. Infection of the urinary tract was ruled out by analysis and culture of a catheter specimen of urine.

Analysis of the data revealed no correlation between the number of deliveries, the difficulties of labor and the degree of incontinence, which was arbitrarily expressed on a basis of + to ++++. The percentile distribution of the various degrees of incontinence among the 95 patients studied is given in Table 2. The findings of the pelvic examination could not be correlated with the severity of the incontinence. In many cases large cystoceles, uterine prolapse or extensive perineal tears were associated with only mild leakage of urine, whereas patients with marked incontinence had surprisingly good pelvic structures. The control group amply demonstrated that urinary continence need not be impaired by post-partum changes affecting the pelvis and perineum.

Table 2. *Distribution of the Degree of Exertional Incontinence.*

Degree of Incontinence*	Percentage of Cases
+	27
++	30
+++	26
++++	17

* + = loss of a few drops of urine; ++++ loss of entire bladder contents; and ++ and +++ = intermediary degrees of incontinence.

The cystometrograms, the cystograms and the cystoscopic findings were similar in both groups and revealed no specific criteria diagnostic of exertional incontinence. A properly functioning internal sphincter was demonstrable even in patients with marked incontinence.

The urethrograms revealed all sorts of urethral configurations without establishing an x-ray picture typical of exertional incontinence. The width and length of the urethra, its course in relation to the posterior surface of the symphysis pubis and its mobility had no demonstrable relation to urinary control (Figs. 1–3).

Discussion

The belief that exertional incontinence is caused by obstetric trauma is time-honored and has never been seriously questioned. This is understandable because the majority of women who suffer from it have borne children and because some of them

*From the Department of Urology, Beth Israel Hospital.
Read at a meeting of the New England Section of the American Urological Association, Boston, April 26, 1945.

†Assistant in urology, Harvard Medical School; instructor in urology, Tufts College Medical School; junior visiting urologist, Beth Israel Hospital.

developed this symptom shortly after childbirth (Table 3).

A good deal of evidence, however, can be cited against the assumption that exertional incontinence

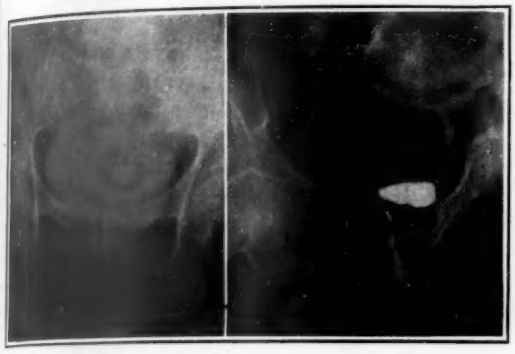

FIGURE 1.

This patient had a reversed urethral curve. She had never been pregnant and had had no incontinence.

invariably results from obstetric trauma. Typical exertional incontinence may occur without pregnancy or pelvic delivery, as shown by the case histories of the 5 nulliparas and 2 patients delivered by cesarean section. Trauma is therefore not a prerequisite for the development of urinary incontinence. The symptom complex, moreover, lacks the clinical attributes of a so-called "traumatic lesion." The sequelae of trauma are usually in direct proportion to the extent of injury. Thus, the greater the injury, the more widespread are the effects. Furthermore, there is a definite time relation between trauma and the ensuing symptoms, which establishes their causal relation. This is not so with exertional incontinence. The extent of obstetric damage to the pelvic and perineal structures cannot be correlated with the degree of urinary leakage. Moreover, in the average case nine years are found to have elapsed before the patient first noted loss of urine on coughing or sneezing. Many women do not become aware of poor urinary control until fifteen to twenty years after the birth of the last child, or indeed not until the menopause. This long time interval between cause and effect is not in keeping with experience in traumatic lesions elsewhere in the body and suggests strongly that the two are not causally related.

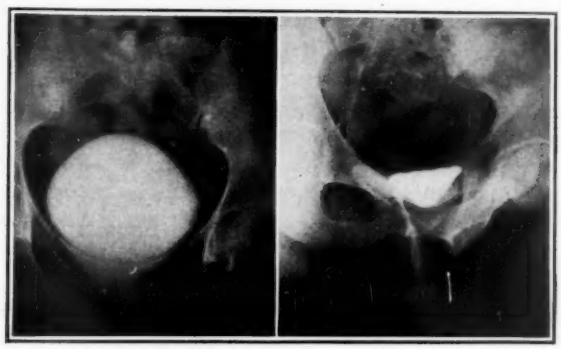

FIGURE 3.

This patient had prolapse of the uterus and a cystocele but no incontinence.

FIGURE 2.

This patient had a large cystocele. There was a wide urethra and slight "funnelling" of the internal sphincter, but no incontinence.

TABLE 3. *The Onset of Incontinence in Relation to Parturition (90 Cases).*

Interval between Childbirth and Onset of Incontinence	Percentage of Cases
yr.	
Less than 1	26
1-4	10
5-9	22
10-14	16
15-19	17
20-24	4
25 and over	5

A urethral lesion pathognomonic of exertional incontinence, moreover, has proved to be elusive. Evidence of its existence has been rather involved and indirect. Considerable interest has recently been evoked by Kennedy's[1-4] suggestion that fixation of a middle urethral sphincter by scar tissue causes exertional incontinence. No such specific lesion or deformity, however, was found on careful study among the 95 patients of this series. Furthermore, the sphincter to which Kennedy refers has never been demonstrated anatomically, and it is doubtful that one thus situated could be of any great

importance to urinary continence. More than two thirds of the female urethra can be safely excised — as it usually is for carcinoma of the urethra — without causing loss of urinary control,[5, 6] showing that the normal mechanism responsible for continence does not reside in the distal two thirds of the urethra.

The only significant urethral sphincter, according to recent investigations, consists of the fibers of the detrusor at the bladder outlet or neck.[7–10] This sphincter is an integral part of the detrusor, and its behavior is entirely dependent on it, as was shown by the studies of Denny-Brown and Robertson.[11, 12] These authors have clearly demonstrated that no independent relaxation of the internal vesical sphincter can occur without corresponding contraction of the detrusor. The bladder neck therefore cannot open even partially unless the detrusor contracts simultaneously. For the escape of urine, to be interrupted, detrusor contractions must be inhibited.

The function of the detrusor in exertional incontinence was investigated in a series of 7 patients. The bladder was filled with Diodrast, and its behavior was observed under the fluoroscope. When micturition was initiated, contraction of the bladder dome was first seen. This was followed by straightening of the lateral walls and centripetal contraction of the entire bladder muscle. When requested to inhibit the urinary stream, control subjects were promptly able to relax the detrusor. Patients with exertional incontinence, on the other hand, invariably accomplished this only with difficulty. The detrusor muscle continued to contract for a variable time after the initial voluntary effort to stop urination, this interval being commensurate with the degree of incontinence. Patients with marked incontinence either inhibited the bladder contractions late during urination or proceeded to complete the micturition.

These observations, although based on the study of a small series of cases, nevertheless emphasize the importance of proper detrusor function in normal micturition and in the presence of urinary incontinence. They suggest that exertional incontinence is due to an acquired irritability of the detrusor that permits minor stimuli, such as are set off by coughing or sneezing, to produce detrusor contractions, with corresponding relaxation of the internal sphincter and urinary leakage. If this assumption is correct, the source of the trouble in exertional incontinence is not in the urethra, its sphincters or supports but in the bladder muscle itself and its internal sphincter. The normal mechanism, which keeps the detrusor relaxed until micturition is initiated or which relaxes it at will when the urinary stream is to be inhibited, is disturbed. The cause of such an acquired detrusor dysfunction, however, must at present be regarded as unknown. In previous reports it was shown that the bladder muscle can be strikingly affected by hormones.[13, 14] The hormonal changes associated with pregnancy and the puerperium profoundly alter bladder capacity and tonus. Hormonal changes occur also at the menopause, when many women first develop exertional incontinence. The precise relation between female hormones and this type of urinary leakage, however, is yet to be established.

The concept of exertional incontinence as a functional bladder disturbance undermines the basis on which the present methods of surgical treatment rest. The various plication operations and the procedures involving the use of fascial slings around the urethra do not attack the problem at its root. By their fixation of the bladder neck they do, however, make it difficult for the internal sphincter to open easily in response to detrusor contractions. It is probably for this reason that overfixation of the bladder neck has often led to inability to void, whereas insufficient fixation has produced only fleeting relief from incontinence.

Summary

No specific urethral lesion was found in a study of 95 patients with exertional incontinence, as compared with 45 control subjects.

The belief that obstetric trauma causes exertional incontinence is critically reviewed, and the conclusion is reached that it is not an etiologic factor.

Evidence is presented that exertional incontinence is due to an acquired dysfunction of the detrusor rather than to anatomic defects.

520 Beacon Street

References

1. Kennedy, W. T. Incontinence of urine in female: some functional observations of urethra illustrated by roentgenograms. *Am. J. Obst. & Gynec.* **33**:19-29, 1937.
2. *Idem.* Incontinence of urine in female: urethral sphincter mechanism: damage of function and restoration of control. *Am. J. Obst. & Gynec.* **34**:576-589, 1937.
3. *Idem.* Incontinence of urine in female: study of urethral sphincter under hydrostatic pressure with roentgenograms — sphincter mechanism, loss of control, restoration. *New York State J. Med.* **38**: 256-261, 1938.
4. *Idem.* Urinary incontinence relieved by restoration and maintenance of normal position of urethra. *Am. J. Obst. & Gynec.* **41**:16-28, 1941.
5. Watson, E. M. Carcinoma of female urethra. *J. Urol.* **35**:651-663, 1936.
6. Lazarus, J. A., and Schneider, A. D. Primary carcinoma of female urethra treated by complete extirpation of urethra. *J. Urol.* **35**: 235-240, 1936.
7. von Lüdinghausen, H. J. H. Die anatomischen Grundlagen des Verschlussmechanismus der weiblichen Harnblase. *Ztschr. f. Anat. u. Entwcklngsgesch.* **97**:757-766, 1932.
8. Van Duzen, R. E. Neurogenic disturbances of vesical function. *J. Urol.* **37**:156-165, 1937.
9. Dragonas, E. Study of functioning of musculature of bladder and mechanism of opening of neck in normal micturition. *Arch. urol. de la Clin. de Necker* **7**:17-46, 1933.
10. Griffiths, J. Observations upon urinary bladder and urethra. *J. Anat. & Physiol.* **29**:254-275, 1895.
11. Denny-Brown, D., and Robertson, E. G. On physiology of micturition. *Brain* **56**:149-190, 1933. State of bladder and its sphincters in complete transverse lesions of spinal cord and cauda equina. *Brain* **56**:397-463, 1933.
12. Denny-Brown, D. E. Nervous disturbances of vesical sphincter. *New Eng. J. Med.* **215**:647-652, 1936.
13. Muellner, S. R. Physiological bladder changes during pregnancy and puerperium. *J. Urol.* **41**:691-695, 1939.
14. Muellner, S. R., and Hamilton, J. B. Effect of testosterone propionate on tonus of urinary bladder. *J. Urol.* **52**:139-148, 1944.

PYOGENIC LIVER ABSCESS

Review of the Literature and Report of a Case Successfully Treated by Operation and Penicillin

LIEUTENANT COLONEL J. EDWARD FLYNN, M.C., A.U.S.*

PYOGENIC abscesses of the liver have caused death in at least half the patients afflicted with this disease, the mortality having ranged from 50 per cent, in Eliason's[1] series, to 95 per cent, reported by Brütt.[2] Since chemotherapy has proved to be of great value in many diseases caused by pyogenic organisms, it seems logical that one of these drugs might be useful in cases of pyogenic liver abscess, and a case so treated is reported.

ETIOLOGY

There are five ways in which pyogenic organisms invade the liver. First, they may travel through the portal vein from regions drained by it. Suppurative appendicitis, complicated by pylephlebitis, is one of the most frequent causes. In Ochsner's[3] collected series of 575 cases, appendicitis was the source in 197 (24 per cent). In the 47 cases of Ochsner's own series, however, appendicitis was the source in 11 per cent. The latter is probably a more accurate figure, because in a series of 231 cases reported by Baerensprung,[4] Rothenberg and Linder[5] and Keefer[6] appendicitis was the source in 13 per cent. Ulcerations of the stomach or bowel and diseases of the rectum, spleen and pancreas frequently cause hepatic abscess. In Ochsner's collected series, they constituted 10 per cent of all causes. Second, blood-borne infections may be transmitted through the hepatic artery. Osteomyelitis, furunculosis, ulcerative endocarditis, acute infections of the upper respiratory tract or pyemia from any source may be the cause. In Ochsner's collected series, 13 per cent were due to blood-borne infections. Third, there may be direct extension from a contiguous infection. A diseased gall bladder was the source in 14 per cent of Ochsner's collected series. Subphrenic abscess, empyema, nephritic or perinephritic abscesses and gastric and duodenal ulcers are less frequently the cause. Lastly, trauma may be the cause. This was so in 3 per cent of Ochsner's collected series. There may be a penetrating wound from a bullet or some other agent or a subcutaneous injury to the liver, producing traumatic changes in it and permitting the bacteria always present in this organ to grow.

Cases of pyogenic liver abscess in which the source cannot be determined form a relatively large group. In Ochsner's collected series, 17 per cent of the cases were of this type. In his own series of 47 cases, the incidence was 59 per cent. Such cases, Beaver[7] believes, are due to lesions draining into the portal vein that are so small that they cannot be localized clinically, whereas Rothenberg and Linder[5] think that they are the result of hematogenous infections, secondary to small, inconspicuous distant foci. A case that falls into this group and in which pyogenic infection was due to nonhemolytic streptococci is presented below.

*Instructor in surgery, Tufts College Medical School, and assistant to visiting surgeons, Second Surgical Service, Boston City Hospital (on leave of absence).

BACTERIOLOGY

The organisms that cause pyogenic abscesses of the liver are generally sensitive to the sulfonamides or to penicillin. The organisms most frequently found are colon bacilli, streptococci, staphylococci or a combination of two or all of these. In Ochsner's collected series, the responsible organisms were colon bacilli in 31 per cent of the cases, streptococci in 21 per cent, staphylococci in 17 per cent, and two or more of these three in 17 per cent. Organisms that are rarely found are *Clostridium perfringens*, *Pseudomonas pyocyaneus*, *Eberthella typhosa*, Leptothrix,[8] Streptothrix,[9] Salmonella,[10] spirochetes[11] and gonococci.[12] Pus in pyogenic abscesses of the liver is frequently sterile. Rothenberg and Linder[5] found sterile pus in 46 per cent of their series, whereas Elsberg[13] reported it positive in 60 per cent of his series.

PATHOGENESIS AND PATHOLOGY

It has been mentioned that pyogenic hepatic abscesses may develop by extension of infection through the portal vein, through hepatic arteries as metastatic abscesses, by direct extension from a nearby organ or by trauma. Munro[14] has stressed the lymphatic vessels, but most investigators believe that infection of the liver rarely if ever occurs by this route. The retroperitoneal route from the appendix to the liver has been mentioned by Korte,[15] Loison[16] and Walter-Sallis.[17]

The most frequent source is considered by most investigators to be suppurative appendicitis. The course of the infection is easy to follow as it proceeds from inflammation of the appendix to thrombophlebitis of the vessels in the appendix wall and thence to progressive phlebitis of the appendiceal, ileocecal, superior mesenteric and portal veins. A septic embolus can, however, be detached from the thrombus in the appendiceal region. Most hepatic abscesses that follow appendicitis are multiple and involve the right lobe of the liver. The existence of two currents of blood in the portal vein, one from the superior mesenteric vein, which leads to the right lobe of the liver, and one from the inferior mesenteric and splenic veins, which lead to the left lobe, has been demonstrated by Sérégé,[18] Glénard,[19] Bartlett, Corper and Long[20] and Copher and Dick.[21]

From the pathological standpoint, the first con-

sideration is the pylephlebitis. The infection in most cases invades the vein from without, the phlebitis being preceded by inflammation in the wall of the appendix. The inflammatory process may extend along the course of the vein, so that phlebitis has every facility for extension. The wall of the vein is infiltrated with leukocytes. Thrombosis rapidly occurs. The clot softens and mingles with the pus from the vessel wall. The inflammatory process may weaken the wall, with subsequent dilation of the vein, which may perforate, permitting the escape of blood and pus into the hepatic tissue. In other cases the inflammation is carried to the wall of the vein, with the early formation of a septic thrombus. Septic emboli may be carried through the portal system and produce hepatic abscesses.

Grossly the liver is enlarged. The abscesses are usually multiple; they vary in diameter from 0.5 mm. to 1.0 cm. and are generally confined to some particular section of the liver, the right lobe being involved oftener than the left. The multiple abscesses give the liver a mottled appearance. Small abscesses may coalesce, forming larger ones, and large solitary abscesses may become chronic. Beaver[7] has described granulomatous abscesses of the liver.

The lung or pleural cavity may be involved by a spread of infection through the diaphragm, or rarely by a septic embolus. Usually a solitary abscess in the right lobe of the liver is the cause of pleuropulmonary complications. In Ochsner's[3] collected series, pneumonia was a complication in 11 per cent of the cases, lung abscesses were present in 9 per cent, and empyema was found in 4 per cent.

Peritonitis may occur as the result of rupture of the abscess into the peritoneal cavity or following transperitoneal drainage of the abscess. In Keefer's[6] series, peritonitis complicated liver abscess in 8 per cent of the cases. Subphrenic abscess was found as a complication in 6 per cent.

If the hepatic abscesses are due to pyemia, abscesses may also be found in the lungs, kidneys, spleen and brain.

Unusual complications have been reported by some investigators. Rupture of a hepatic abscess into the pericardium has been reported by Keefer[6] and Azar,[22] into the abdominal wall, by Huard et al.,[23] into the vena cava, by McKnight[24] and into the thoracic duct by Nieweg.[25] The occurrence of a pulmonary infarct has been cited by Keefer.[6]

Symptoms and Signs

The usual systemic symptoms are fever, chills, pain and profuse sweating. Less often malaise, anorexia, loss of weight, weakness, nausea and vomiting occur. Fever may be continuous, intermittent or remittent. With acute suppurative appendicitis complicated by pylephlebitis and multiple abscesses of the liver, fever is of the so-called "picket-fence" type and is generally accompanied by daily chills. Rothenberg and Linder[5] observed that daily chills occurred frequently with multiple hepatic abscesses but were rare with a single abscess. As far back as 1897 Bryant[26] called attention to the significance of chills and fever during the course of appendicitis. Brown,[27] Gerster,[28] Babler,[29] Thalhimer,[30] Eliason,[1] Ochsner et al.[31] and others have emphasized this point.

Pain is felt in the right upper quadrant of the abdomen and is usually constant. In Ochsner's collected series, pain was present in 91 per cent of the cases. Dixon and Murphy[32] observed radiation of pain from the right hypochondrium to the right shoulder, probably owing to irritation of the phrenic nerve. Rothenberg and Linder[5] believe that irritation of the phrenic nerve seldom occurs, but state that over one third of patients complain of pain in the right lower chest on inspiration, indicating a diaphragmatic pleurisy. Intercostal pain may occur, as described by Rothenberg and Linder, Eliason[1] and Alessandri.[33] Weakness, anorexia and loss of weight are frequent. Nausea and vomiting seldom occur and were present in less than one third of Ochsner's collected cases.

Tenderness over the liver area and hepatic enlargement are frequent local signs. Rothenberg and Linder[5] found tenderness in all their cases and hepatic enlargement in 92 per cent.

Jaundice occurs infrequently and is usually a late serious sign. Jaundice occurred in 8 per cent of Rothenberg and Linder's cases and in 25 per cent of Ochsner's collected series. Ascites is rare.

Laboratory Data

Leukocytosis, with an increase in polymorphonuclear leukocytes, is almost invariably present. The contrast between the moderate leukocytosis accompanying amebic abscess and the greater leukocytosis of pyogenic hepatic abscess has been mentioned by Rogers,[34] Manson-Bahr and Willoughby[35] and Ochsner and DeBakey.[36] The leukocytosis in acute cases is about twice as high as that in chronic cases.

Urinalysis is usually negative, but with jaundice bile is found in the urine, and with marked toxemia there is albuminuria.

Ochsner[3] believes that roentgenography is one of the most significant and reliable aids in the diagnosis of liver abscess. The elevation and immobility of the diaphragm, usually on the right, as seen by x-ray are of high diagnostic value according to Pancoast,[37] Dickinson,[38] Love[39] and Oschner.[3] Granger[40] and Ochsner and DeBakey[36, 41–43] have observed that in subphrenic abscess complicating liver abscess there is obliteration of the cardiophrenic angle in the anteroposterior view and of the anterior costophrenic angle in the lateral view, whereas in subphrenic abscess due to other causes there is obliteration of the costophrenic angle in

the anteroposterior view and of the posterior costophrenic angle in the lateral view. Miles[44] showed that a gastrointestinal series in the presence of an abscess in the left lobe of the liver presents a deformity in which the lesser curvature of the stomach assumes a crescentic shape and the cardia and duodenal cap are displaced.

Differential Diagnosis

It is often possible to differentiate multiple and single abscesses of the liver. The former have an acute, sudden onset and are usually secondary to pylephlebitis and suppurative appendicitis, whereas single pyogenic abscesses generally have an insidious onset and in many cases it is impossible to determine the source. Fever, chills and leukocytosis are more prominent with multiple than with single abscesses. Amebic abscesses of the liver usually have an insidious onset. The temperature is only slightly increased, chills rarely occur, and *Endamoeba histolytica* may be found in the stools.

Intrahepatic — toxic or infectious — jaundice is accompanied by damage to the parenchymal liver tissue. The van den Bergh reaction is generally direct.[45] There may be no pain and little or no fever, and the onset is usually insidious.

Acute cholecystitis and empyema of the gall bladder must frequently be considered. In cases in which there are definite pathologic changes in the right upper quadrant of the abdomen, a conscientious perusal of the past history may be a great aid in establishing a diagnosis of acute cholecystitis. If there have been repeated attacks after the ingestion of fatty or fried foods or repeated attacks of colic of the type associated with gallstones, it is reasonable to assume that the gall bladder is the seat of the trouble. Nausea and vomiting are frequent. Definite localized tenderness and rigidity of the right rectus muscle in the upper quadrant of the abdomen can be demonstrated.

Exploratory aspiration of the liver for detection of pus in pyogenic hepatic abscess is a dangerous procedure and should be condemned because of the danger of contaminating the pleura or peritoneum. This condition can be readily diagnosed by other methods.

Prognosis

In the past, the prognosis with hepatic abscess has been extremely poor. Dieulafoy[46] in 1898 held that hepatic abscess following appendicitis was invariably fatal. Thompson[47] and Gerster[28] a few years later wrote of the seriousness of the condition. In 1914, Petrén[48] reported a mortality of 93 per cent. Later, Brütt[2] recorded one of 95 per cent, and in 1932 Otschkin[49] gave one of 80 per cent. Ochsner[3] clearly demonstrated that the mortality is much greater with multiple abscesses (95 per cent) than with single ones (37 per cent).

Obviously the prognosis in cases of pyogenic hepatic abscess has been extremely grave, and it is of interest that the mortality rate has changed little during the last fifty years. With chemotherapy, however, it should be greatly reduced.

Treatment

As a prophylactic measure, in cases of appendicitis with a history of preoperative chills, the appendiceal, ileocolic, superior mesenteric and portal veins should be examined during operation, as suggested by Melchior[50] and Thalheimer.[30] This may reveal a thrombosis, prompt treatment of which may prevent the development of hepatic pyogenic abscess. It seems likely that chemotherapy will also prove to have great prophylactic value.

The treatment of pylephlebitis is surgical. Melchior[51] reported 13 cases in which ligation of the ileocolic vein was performed. Eight of these patients had ligation at the time of appendectomy or drainage, and none died. In the other 5 cases ligation was performed after the primary operation, and 1 patient died. In 1935, Stewart–Wallace[52] reported 15 cases in which primary ligation was performed. Two patients died from peritonitis. He also reported 6 cases in which the superior mesenteric vein was ligated; 4 patients recovered. Stewart–Wallace concluded that it is possible to ligate the superior mesenteric vein at or below the levels of the transverse mesocolon and the third part of the duodenum without the development of bowel necrosis.

Incision and drainage is the proper treatment of solitary pyogenic hepatic abscess. Ochsner[3] stresses the importance of using the type of drainage that avoids the possibility of contamination of the pleura or peritoneum.

An extraserous anterior or posterior approach may be made, depending on the location of the abscess. With an abscess on the anterior or anteroinferior surface of the liver, a procedure similar to that described by Clairmont and Meyer[53] for drainage of subphrenic abscesses should be used. A skin incision is made anteriorly just beneath and parallel to the costal margin. The external and internal oblique muscles and transversalis fascia are cut. The parietal peritoneum is carefully dissected from the inferior surface of the diaphragm. If the abscess is reached, it is drained extraperitoneally at this stage. If it is not reached extraperitoneally, the visceral and parietal surfaces of the peritoneum are made to adhere by packing the area with gauze impregnated with some irritative substance, and drainage is later performed through this area.

If there is no sign of localization of the abscess in the anterior abdominal region, the retroperitoneal approach described by Ochsner[43, 54] is used. A skin incision is made directly over the twelfth rib, which is resected subperiosteally. A transverse incision is made through the resected bed of the

twelfth rib and the serratus posterior inferior muscle at the level of the spinous process of the first lumbar vertebra. The pleura at the costophrenic angle never extends below the spinous process of the first lumbar vertebra. The retroperitoneal space is entered between the upper pole of the kidney and the inferior surface of the liver. The parietal peritoneum is dissected from the undersurface of the diaphragm and the liver abscess is drained extraserously. The importance of an extraserous approach is stressed by Ochsner,[3] who cites a mortality of 33 per cent with an extraserous approach, one of 67 per cent with a transpleural approach and one of 73 per cent with a transperitoneal approach.

Chemotherapy should play a significant role in the decrease in mortality with multiple pyogenic abscesses of the liver. If operation is performed it is important to culture the organisms and test their sensitivity to penicillin and to sulfadiazine.

* * *

A case of multiple pyogenic abscesses of the liver, due to nonhemolytic streptococci, with no demonstrable focus, is presented below. The organism was sensitive to penicillin. The patient made a complete recovery. No similar case can be found in the literature.

Case Report

The patient was a 23-year-old American soldier, born in Italy. The first symptom was a dull pain in the right upper quadrant of the abdomen, which lasted for 30 minutes. The pain recurred in 2 days, and the patient was admitted to a station hospital. The pain was intermittent and dull and persisted for 3 days. On the 3rd day after the onset of symptoms, the patient became jaundiced. On the 8th day, chills and a temperature of 101°F. developed, persisting for 3 days. On the 14th day, a temperature of 101°F. and chills recurred and lasted for 2 days. On the 21st day, pain recurred in the right upper quadrant of the abdomen. The patient vomited twice and had chills and a temperature of 102°F. Pain persisted for 1 day. On the 26th day, pain, fever, chills and vomiting recurred and lasted for 1 day. Jaundice and clay-colored stools persisted for 3 weeks. A diagnosis of catarrhal jaundice was made. After 5 weeks the patient was transferred to a convalescent hospital.

The patient was symptom-free for 3 days while in the convalescent hospital. On the 41st day after the onset of symptoms, the fifth attack of pain occurred. Pain was felt in the right and left upper quadrants of the abdomen and radiated to the right shoulder. The patient vomited once and jaundice recurred for the second time. There were chills and a temperature of 103°F. On physical examination there was tenderness and spasm over the right upper quadrant of the abdomen. The white-cell count was 16,500. Pain and jaundice persisted for 1 week.

The patient was symptom-free for the next 10 days. Pain in the right upper quadrant recurred for the sixth time, and jaundice for the third time. The liver edge was palpated 2 fingerbreadths below the costal margin. There were tenderness and spasm in the right upper quadrant. A diagnosis of probable empyema of the gall bladder was made, and the patient was prepared for surgery.

Operation was performed under spinal anesthesia, and the abdomen was opened. The liver was enlarged, with many white areas varying from 1 mm. to 5 cm. in diameter. A large, fluctuant mass measuring 5 cm. in diameter was seen. Pus was aspirated from the abscess and a biopsy of the liver edge was performed. The gall bladder was distended, and grayish fluid was aspirated from it. The common duct was not dilated. A cholecystostomy was performed, and a cigarette drain was inserted in the right subhepatic region. The wound was closed in layers except for the drain. A culture of pus aspirated from the abscess at operation showed microaerophilic nonhemolytic streptococci. The organisms were sensitive to penicillin, the zone of inhibition being 18 mm. in diameter.

The pathological report was as follows: The gross specimen consists of a section of the liver measuring 5 by 3 by 2 cm. A gray area of pin-point size is seen in the yellow gray surface. Lobulation cannot be made out.

Microscopical examination shows that about half the normal architecture has been destroyed by an acute inflammatory process in and about the bile ducts, with necrosis of the adjacent liver cords and abscess formation. In addition to infiltration by polymorphonuclear leukocytes, there are a considerable number of eosinophils in the inflammatory reaction. There is hyperplasia of duct epithelium and early fibroplastic replacement of destroyed parenchyma. Diagnosis: acute cholangitis, with abscess formation.

Urinalysis was negative except during the periods when the patient was having pain and jaundice, when bile and albumin were found. On the day of operation the urine was amber, the specific gravity was 1.010, and there was a ++ test for albumin. There was an acid reaction, and no sugar and the test for bile was strongly positive.

The white-cell count during the acute attacks ranged from 10,500 to 21,000, with 79 per cent segmented neutrophils, 12 per cent lymphocytes, 6 per cent stab forms, 2 per cent monocytes and 1 per cent eosinophils. The red-cell count during the acute attacks ran about 3,100,000, with a hemoglobin of 78 per cent. Multiple smears showed no malarial parasites.

The icteric index during the phases of jaundice ranged from 11 to 43. The prothrombin time on the day before operation was 4 minutes for the patient and 3 minutes for the control. Three weeks postoperatively it was 16 and 6½ minutes, respectively, and 5 weeks postoperatively it was 5.5 and 4 minutes, respectively.

Three weeks preoperatively, the van den Bergh reaction was indirect, with 0.3 mg. per 100 cc. Three weeks postoperatively, it was again indirect, with 2.4 mg. and 4 weeks postoperatively it was indirect, with 2.1 mg.

Serologic tests for syphilis, tularemia, undulant fever and typhoid fever were negative. Dark-field examination of the blood was negative. Multiple stool examinations were negative for bile, occult blood, parasites and ova.

Blood cultures taken 4 weeks and 1 week preoperatively showed no growth. Culture of bile from the cholecystostomy showed nonhemolytic streptococci, nonhemolytic staphylococci and gram-negative rods not of the enteric group. Many x-ray films of the chest and abdomen showed no abnormality.

An electrocardiograph taken 3 weeks preoperatively showed sinus tachycardia. Low amplitude and low T waves were observed, findings similar to those in coronary disease, early pericarditis and systemic infection of long standing.

The patient was given 20,000 units of penicillin every 3 hours for 3 weeks postoperatively. Intravenous therapy was given for 4 days, and oral feeding was then begun. Five days postoperatively the temperature became normal and remained so. The cholecystostomy tube fell out on the 5th day. The wound was entirely healed 16 days after operation, when the patient was permitted out of bed. He was discharged 6 weeks after operation. A check-up examination 5 months after operation was negative. The patient will be followed indefinitely, especially for signs of biliary cirrhosis.

One and a half years postoperatively, the patient reported that he was well.

Summary

Pyogenic hepatic abscess has caused a mortality of 50 to 95 per cent in patients afflicted with this disease.

Since the infection is usually caused by an organism that is sensitive to the sulfonamides or penicillin, chemotherapy should cause a great decrease in the mortality, but early diagnosis and proper operative procedures are also important.

A case of multiple pyogenic hepatic abscesses is presented. This case may be the forerunner of many others in which the patients survive when treated properly with penicillin or sulfadiazine. Etiologically, this patient belonged to the so-called "idiopathic group," in which the source of the infection cannot be determined. The organism was a nonhemolytic streptococcus sensitive to penicillin. Recovery followed penicillin therapy of three weeks' duration.

References

1. Eliason, E. L. Pylephlebitis and liver abscess following appendicitis. *Surg., Gynec. & Obst.* **42**:510-522, 1926.
2. Britt. Zur Chirurgie der Pylephlebitis nach Appendicitis. *Zentralbl. f. Chir.* **1**:610-614, 1922.
3. Ochsner, A., DeBakey, M., and Murray, S. Pyogenic abscess of liver: analysis of 47 cases with review of literature. *Am. J. Surg.* **40**:292-319, 1938.
4. Baerensprung, C. Der Leberabscess nach Kopfverletzungen. *Arch. f. klin. Chir.* **18**:586-593, 1875.
5. Rothenberg, R. E., and Linder, U. Single pyogenic liver abscesses: study of 24 cases. *Surg., Gynec. & Obst.* **59**:31-41, 1934.
6. Keefer, C. S. Liver abscess: review of 85 cases. *New Eng. J. Med.* **211**:21-24, 1934.
7. Beaver, D. C. Granulomatous abscess of liver of pyogenic origin. *Am. J. Path.* **7**:259-276, 1931.
8. Harris, P. N. Multiple liver abscesses caused by leptothrix, with review of leptothrical infection. *Am. J. Path.* **9**:71-90, 1933.
9. Bloomfield, A., and Bayne-Jones, S. Case of abscess of liver due to Streptothrix. *Bull. Johns Hopkins Hosp.* **26**:230-233, 1915.
10. Lee, H. L. Salmonella enteritidis in liver abscess: report of case. *Chinese M. J.* **49**:577-580, 1935.
11. Ecker, E. E., and Lynch, J. Spirochetal abscess of liver. *Arch. Path.* **20**:253-255, 1935.
12. Baker, B. M., Jr., and Carter, E. P. Three instances of unusual gonococcal infection. *Bull. Johns Hopkins Hosp.* **50**:57-75, 1932.
13. Elsberg, C. A. Solitary abscess of liver. *Ann. Surg.* **44**:217-235, 1906.
14. Munro, J. C. Lymphatic and hepatic infection secondary to appendicitis. *Ann. Surg.* **42**:692-734, 1905.
15. Korte. *Beitrage zur Chirurgie der Gallenwege und der Leber.* Berlin, 1905.
16. Loison, E. Des suppurations intra et péri-hépatiques d'origine typhlo-appendiculaire. *Rev. de chir.* **21**:522-539, 1900.
17. Walter-Sallis, J. Le foie dans l'appendicite. *Rev. de chir.* **49**:181-201 and 649-688, 1914.
18. Sérégé, H. Contribution à l'étude de la circulation du sang porte dans le foie et des localisations lobaires hépatiques. *J. de méd. de Bordeaux* **31**:271-273 and 312-314, 1901. Etude sur l'indépendence anatomique et physiologique des lobes du foie. *J. de méd. de Bordeaux* **32**: 327-329 and 357-359, 1902.
19. Glénard, F. Note sur les localisations lobaires hépatique. *Bull et mém. Soc. méd. d. hôp de Paris* (Series 3) **18**:386-397, 1901.
20. Bartlett, F. K., Corper, H. J., and Long, E. R. Independence of lobes of liver. *Am. J. Physiol.* **35**:36-50, 1914.
21. Copher, G. H., and Dick, B. M. "Stream line" phenomena in portal vein and selective distribution of portal blood in liver. *Arch. Surg.* **17**:408-419, 1928.
22. Azar, A. J. Large spontaneous abscess of liver with rupture into pericardium. *Illinois M. J.* **61**:428, 1932.
23. Huard, P., Renucci, N., and Huynh-Doi. Les abcès du foie non amibiens. *Bull. Soc. méd.-chir. de l'Indochine* **13**:849-873, 1935.
24. McKnight, R. B. Rupture of hepatic abscess into abdominal cavity and inferior vena cava. *J. A. M. A.* **90**:1929, 1928.
25. Nieweg, G. A. Abscess of liver and right psoas muscle secondary to gangrenous appendix. *U. S. Vet. Bur. M. Bull.* **5**:58-60, 1929.
26. Bryant, J. H. Suppurative pylephlebitis. *Guy's Hosp. Rep.* **54**: 77-89, 1897.
27. Brown, W. L. Pylephlebitis. *St. Bartholomew's Hosp. Rep.* **37**:53-180, 1901.
28. Gerster, A. G. On septic thrombosis of roots of portal vein in appendicitis and on pylephlebitis, together with some remarks on "peritoneal sepsis." *M. Rec.* **63**:1005-1015, 1903.
29. Babler, E. A. Pylephlebitis complicating appendicitis. *Ann. Surg.* **61**: 589-596, 1915.
30. Thalhimer, W. Chills occurring early in appendicitis before operation and their indication of operable stage of pylephlebitis. *Arch. Surg.* **8**:658-668, 1924.
31. Ochsner, A., Gage, I. M., and Garside, E. Intra-abdominal postoperative complications of appendicitis. *Ann. Surg.* **91**:544-572, 1930.
32. Dixon, C. F., and Murphy, G. T. Primary idiopathic abscess of liver. *Surg., Gynec. & Obst.* **54**:20-25, 1932.
33. Alessandri, R. Surgery of liver. *Nelson's New Loose-Leaf Surgery.* Vol. 5. New York: Thomas Nelson & Sons. Pp. 531-608.
34. Rogers, L. Amoebic liver abscess: its pathology, prevention, and cure. *Lancet* **1**:463-469, 1922.
35. Manson-Bahr, P. H., and Willoughby, H. On leucocyte count in liver abscess. *Tr. Roy. Soc. Trop. Med. & Hyg.* **22**:465-467, 1929.
36. Ochsner, A., and DeBakey, M. Liver abscess: amebic abscess: analysis of 73 cases. *Am. J. Surg.* **29**:173-194, 1935.
37. Pancoast, H. K. Roentgenological diagnosis of liver abscess with or without subdiaphragmatic abscess. *Am. J. Roentgenol.* **16**: 303-320, 1926.
38. Dickinson, J. C. Radiographic findings in hepatic abscess, amebic in type. *Radiology* **4**:273-278, 1925.
39. Love, R. T. M. Amebic abscess of liver. *Brit. M. J.* **1**:696, 1918.
40. Granger, A. Radiological signs of subdiaphragmatic abscess. *New Orleans M. & S. J.* **82**:748-751, 1930.
41. Ochsner, A., and DeBakey, M. Diagnosis and treatment of amebic abscess of liver: study based on 4,484 collected and personal cases. *Am. J. Digest. Dis. & Nutrition* **2**:47-51, 1935.
42. DeBakey, M., and Ochsner, A. Diagnosis and treatment of amebic hepatic abscess. *New Orleans M. & S. J.* **88**:347-352, 1935.
43. Ochsner, A., and DeBakey, M. Subphrenic abscess: collective review and 3,608 collected and personal cases. *Internat. Abstr. Surg.* **66**: 426-438, 1938.
44. Miles, J. M. Roentgenological diagnosis of abscess of concave surface of liver. *Am. J. Roentgenol.* **35**:65-69, 1936.
45. Walter, W., and Friedell, M. T. *Christopher's Textbook of Surgery.* Third edition. 1291 pp. Philadelphia: W. B. Saunders Company, 1942.
46. Dieulafoy. Le foie appendiculaire: abcès du foie consecutifs à l'appendicite. *Semaine méd.* **18**:449-452, 1898.
47. Thompson, R. Pylephlebitis and liver abscess following appendicitis. *Med. & Surg. Rep., Boston City Hospital* **13**:140-160, 1902.
48. Petrén, G. Ueber Leberabscesse als Komplikation zu akuter Appendicitis. *Beitr. z. Klin. Chir.* **94**:225-267, 1914.
49. Otschkin, A. D. Zur Klinik der Thrombose der Mesenterialvenen und der Pfortader bei Appendicitis. *Arch. f. klin. Chir.* **171**:758-774, 1932.
50. Melchior, E. Ueber erfolgreiche sekundäre Ausführung der Braunschen Operation (Ligatur der Vena ileo-colica) bei postappendicitischer Thrombophlebitis meseraica. *Zentralbl. f. Chir.* **54**:3083, 1927.
51. *Idem.* Zur Kenntnis der portalen Infektion im Gefolge der Appendicitis und ihrer Behandlung mittels Venenligatur. *Beitr. z. klin. Chir.* **142**:795-823, 1928.
52. Stewart-Wallace, A. M. Pylephlebitis complicating appendicitis and its treatment by ligature of mesenteric veins. *Brit. J. Surg.* **23**: 362-376, 1935.
53. Clairmont, P., and Meyer, M. Erfahrungen über die Behandlung der Appendicitis. *Acta chir. Scandinav.* **60**:55-134, 1926.
54. Ochsner, A., and Graves, A. Subphrenic abscess: analysis of 3,372 collected and personal cases. *Ann. Surg.* **98**:961-990, 1933.

MEDICAL PROGRESS

ABDOMINAL SURGERY

ARTHUR W. ALLEN, M.D.*

BOSTON

MANY contributions relating to surgical lesions of the abdomen have been reviewed during the past year. It is impossible to refer to all of them in this report, but a large proportion of the most significant are mentioned, and comments are made according to their relation to experience at the Massachusetts General Hospital.

ANESTHESIA

Lemmon and Hager[1] have reported on continuous spinal anesthesia in 2000 cases. They had no deaths and few complications from this method. Of these patients, 3.1 per cent had post-anesthesia headache, 3.3 per cent had urinary retention, and 2.3 per cent had pulmonary complications. Four and one tenth per cent of the patients died from causes unrelated to the anesthesia in an average of about six days. The surgical procedures were mainly abdominal and pelvic but included forty-eight thoracic operations.

This experience is in accord with that of Arrowood and Foldes,[2] who have modified Lemmon's method by a continuous intrathecal novocain drip. The patient is often kept asleep by small doses of Pentothal Sodium given intravenously as an adjunct to the spinal novocain. In this modification the relaxation of the abdominal muscles is continuous and completely satisfactory to the surgeon.

HERNIA

Singleton and Stehouwer[3] report their experience with the fascia-patch method of hernia repair in 129 cases, supplementing a preliminary report on 44 patients made in 1916. They remove a semilunar-shaped piece of fascia lata and suture it to the structures bordering the inguinal canal. They have had recurrences in 3 per cent of indirect, 11 per cent of direct and 18 per cent of ventral and incisional hernias — an average rate of 11 per cent. They point out that 62 per cent of their cases had had previous repairs and that most of these were described as difficult.

Mair[4] makes a preliminary report on the use of a whole-thickness skin transplant in the repair of hernia. The region is prepared with soap and water for three successive days before operation. The incision over the hernia is elliptical, so that a piece of skin measuring 2.5 by 5.0 cm. can be obtained and placed in normal salt solution. The cord is elevated the sac is attended to in the usual manner, and the floor of the canal is narrowed by sutures. The graft is then freed of fat, cut off square at both ends and sutured under the cord to the rectus fascia, Poupart's ligament and the conjoined tendon. The outer end of the graft is split and sutured around the cord to make a new internal ring. Cultures from the graft have occasionally yielded *Staphyloccocus albus*. In spite of this, there were only 2 cases of wound infection in 88 cases, and apparently neither of these caused sloughing of the graft. Mair's experimental work on rabbits and the re-exploration of the region of the graft three months later for some reason in one of his patients enables him to state that skin under these circumstances changes its character to a white fibrous structure. His patients were allowed up on the twentieth postoperative day.

Skinner and Duncan[5] report on one hundred and twenty-six recurrent hernias repaired by them, with four failures, which were due to incorrect selection of procedure, infection, cough and poor tissues. They encountered five types of recurrence, namely, at the internal ring, near the pubis, at the midportion of the canal, as a femoral hernia overlooked at the first operation and as a general bulge below the repair.

Many reports substantiate our belief that when hernias are recurrent or large or the tissues are poor, the fascial-strip method of Gallie and LeMeasurer is the simplest procedure and offers the best opportunity for cure.

APPENDICITIS

Ochsner and Johnston[6] have made a comparative study of the records on patients admitted to the Charity Hospital in New Orleans with appendiceal peritonitis in the years 1933 and 1943. The percentage of ruptured appendices was reduced, but the percentage of cases having received preadmission catharsis remained unchanged. There was a lower mortality and a lower morbidity rate in the more recent group of cases. These authors give credit to chemotherapy, the use of blood and plasma, more logical fluid replacement and delayed wound closure for their better results. They stress the use of delayed operation only in the presence of a palpable mass.

This is in accord with the viewpoint of McPherson and Kinmonth,[7] who have analyzed the records of

*Lecturer in surgery, Harvard Medical School; chief, East Surgical Service, Massachusetts General Hospital.

730 consecutive cases of acute appendicitis admitted to St. Thomas's Hospital in London. They believe that all patients save those presenting a palpable mass should be operated on soon after admission. They recognize that children are exceptions to this rule, and advise modification of this dictum in pregnancy and in the aged. Eight of the 21 fatalities in their series were in patients over sixty years of age.

The above two reports are in accord with the findings of Rogers and Faxon,[8] who studied the appendicitis data from the Massachusetts General Hospital. The rationale of not spreading a localized inflammatory mass by immediate appendectomy is obvious. The abscess can be drained if it develops, and the appendix can be safely removed a few weeks later. Better preoperative and postoperative management has reduced the morbidity and mortality in cases with spreading peritonitis. There still remains an occasional case of this sort in which the patient is too ill for immediate surgery.

Sewell[9] reports on two groups of patients from the same community and attributes a marked improvement in results largely to the use of sulfonamides. He reports the loss of 1 patient in the last consecutive 300 cases of appendicitis treated. Of these, the appendix was gangrenous in 31 cases and was perforated at the time of operation in 33. Sewell believes that the elimination of drains and the change from cat- .t to fine silk have also contributed to the reduction of intestinal obstruction and wound infection.

Obstructive appendicitis is discussed by Bowen.[10] He reviews Wilkie's[11] original concept of this type of appendicitis. It appears that about 14 per cent of all Bowen's cases of acute appendicitis were of the obstructive type. In 38 cases studied for the cause of this condition, a stercorolith was present in 21, kinks and bands in 4, and stenosis in 1, whereas in 12 cases the only etiologic factor seemed to be spasm. Inasmuch as fever develops late and leukocytosis is always present early, there is little excuse for not recognizing this entity before perforation occurs. Since this type of appendicitis accounts for most of the cases of associated peritonitis due to early rupture of the appendix, and therefore for most of the deaths in appendicitis, it is all the more important to have this possibility in mind when acute abdominal symptoms occur.

Knox[12] reviews the literature on primary lymphosarcoma of the appendix and cites another case, making a total of 24 cases so far reported. Of twelve and a half thousand appendectomies performed at St. Luke's Hospital, New York City, since 1910, this was the only case in which such a diagnosis was made.

Biliary System

From the literature since 1900 Dixon and Lichtman[13] have collected 50 cases of congenital absence of the gall bladder, and have added 10 cases from the records of the Mayo Clinic. Fifty-eight per cent of these 60 patients had symptoms of cholecystic disease, 48 per cent had jaundice, and 27 per cent had gallstones in the ducts. Of the patients beyond the age of forty-five, 73 per cent had developed symptoms referable to the biliary tract.

Two case reports of papilloma of the gall bladder are presented by Greenwald.[14] Such patients have the symptoms found in the more frequent pathologic states of the biliary system. The diagnosis is made by roentgen-ray examination after administration of the usual dye for cholecystograms. Palpation of the gall bladder does not reveal the lesion, because the papillomas are soft. Cholecystectomy is recommended. The possibility of malignant degeneration is discussed.

Smith[15] has reported the experience of the Presbyterian Hospital, New York City, with acute cholecystitis for a ten-year period ending in 1941. He concludes that operation should be undertaken as soon as the patient is properly prepared and optimum operating-room facilities are available. The operative mortality increased with the duration of the acute process. Two hundred and twenty-three patients had cholecystectomy, with 8 deaths, a mortality of 4 per cent. Of 103 who had cholecystostomy, 12 died. There was an overall mortality of 7 per cent, which corresponds closely to the mortality of a similar group of patients at the Massachusetts General Hospital.[16]

An excellent discussion on the pitfalls in cholecystectomy has been presented by Simon.[17] Schematic drawings and some photographs taken at operation illustrate the numerous anomalies that may be found in the ducts and blood vessels of the gall-bladder region. The author believes that these variations account for many of the catastrophes occurring during cholecystectomy. He thinks that the surgeon obtains a better opportunity for recognizing the anomalous as well as the normal structures by making the dissection from the fundus of the gall bladder toward the ducts.

Colp[18] has reported the results of repair in 5 cases of traumatic stricture of the hepatic ducts. He used a fenestrated tube placed well up in the right hepatic duct and led into the duodenum. In 1 patient, the tube was brought out through the stomach wall and failed because it had to be removed on the eleventh day after a gastric fistula had developed. One patient was well for four and a half years, 1 for one and a half years, and 1 for two months. The remaining 2 patients have had recurrent chills and fever.

Peterson and Cole[19] treated 3 patients for biliary obstruction due to sclerosing pancreatitis. All had different types of operative procedures to restore the bile flow to the gastrointestinal tract. These authors prefer the Roux type of operation, and have tried to make valvelike infoldings in the segment

of bowel leading away from the liver in an effort to prevent regurgitation of gastrointestinal contents into the hepatic system.

I[20] have described in detail a method of re-establishing continuity between the hepatic ducts and the intestine, based on the Roux principle. Fourteen patients have been operated on by this method so far. In those whose ducts are large and the obstruction is due to pancreatic disorders, the method is simple and easily accomplished. In those who have suffered trauma to the ducts, an end-to-end suture should be made if enough of the duct is available after excising the stricture. Often, however, little of the common hepatic duct is left, and in these cases we believe that our method is particularly applicable. The anastomosis should be made over a removable tube. The chief difficulties have arisen when the tube has come out of the duct too soon. We believe that it should be left in place for three months, in the hope that a permanent communication can be established and the supporting tube eliminated.

Lahey[21] has written an excellent editorial on the subject of injuries to the common and hepatic bile ducts. He stresses the importance of early recognition of the injury and the need for repair by surgeons experienced in this field.

Stomach

The relation between achlorhydria, polyposis and ultimate carcinoma of the stomach has been synchronized by Castle.[22] Olson and Heck[23] studied the data available at the Mayo Clinic and found that only 2 of 63 patients with untreated pernicious anemia developed cancer of the stomach in an average of two years. Thirty-one patients were treated for their anemia, and 6 of these developed cancer of the stomach in an average of five and a half years. Thus, it appears that, with a better life expectancy from pernicious anemia, the ratio of cancer of the stomach in such cases will be much higher than it is in normal persons.

Bisgard and Overmiller[24] believe that radical resection is indicated in perforated cancer of the stomach. They have collected from the literature 100 cases in which the perforation was closed or tamponaded by omentum; 76 of these patients died. In fifteen resections for this complication, there were 2 fatalities. These authors believe that 50 per cent of the cancers of the stomach that perforate are resectable.

Engel[25] advocates the fashioning of a gastric pouch following total gastrectomy for cancer. Since some patients complain of not being able to take food in adequate quantities following total gastrectomy, his concept seems logical. The pouch is constructed by making a long anastomosis between the two limbs of the jejunum, starting just below the esophagojejunostomy. It has long been recognized that a liberal enteroenterostomy between the afferent and efferent jejunal limbs is advantageous. The larger pouch may well be an asset and should be tried.

Gastrojejuncolic fistula has been discussed by Marshall.[26] He advocates a preliminary ileosigmoidostomy to eliminate regurgitation of intestinal contents into the stomach. Later the fistula is resected, preferably with the usual radical stomach resection for duodenal ulcer. In 14 patients, there was 1 death following the first stage.

Ransom[27] reports on 14 cases of gastrojejunocolic fistula treated as follows. Ten patients had the fistulas resected without radical operation for the original duodenal ulcer, with 1 death. There were good results in 4 cases and recurrence of duodenal ulcer symptoms in 5 cases, in 3 of which radical subtotal gastrectomy was performed. Four patients were subjected to radical operation at the time of resection of the fistula, and 1 of these failed to survive.

Duodenum

Perforated duodenal ulcer is again discussed by Graham and Tovee.[28] Of 114 patients admitted in a fifteen-year period to a service of the Toronto General Hospital, 111 were operated on, with 7 fatalities, a mortality of 6 per cent. Three patients not operated on, for reasons that seemed justifiable, died. The ratio of males to females was 107:7. All the patients came from within a twenty-five-mile radius, and all had had recent perforations. The danger of long-distance transportation in such an emergency is stressed. Graham and Tovee urge proper preoperative preparation in patients who arrive in poor condition. All the perforations were closed with omental tabs held in place by three catgut sutures, on the theory that fibrinous sealing is the aim of operation. The age of the patients varied from sixteen to eighty-three years and averaged forty-seven years. These authors are inclined to believe that age is not too important as a factor in mortality. In 34 of 59 cases in which cultures were taken from the peritoneal cavity, no organisms were grown. Drainage of the abdominal cavity was used in only 14 cases. Spinal and Pentothal Sodium anesthesia is preferred. These authors are adamant in insisting on the danger of a more radical procedure than simple closure of the perforation.

Price and Lee[29] closed experimental perforations in dogs' stomachs with omental tabs. All the closures made by omentum with an intact blood supply were successful. In many of the cases in which the tamponade was made with free omental grafts, the grafts became necrotic and leakage occurred.

Gray and Sharpe[30] followed the course of 52 of 62 patients who had been operated on for massive hemorrhage from duodenal ulcer by the Devine exclusion procedure. Five per cent of the patients failed to survive the operation. Forty per cent of

the survivors had later symptoms. The older patients did better than the younger ones, 18 of 21 fifty years of age or under having later symptoms, whereas only 3 of 16 over fifty did so. These authors conclude that this procedure may be justifiable in the older age group. They admit that it is a poor operation for duodenal ulcer, since only 60 per cent of the 52 patients remained symptom-free during the period of observation.

Colp et al.[31] report the comparative immediate results in two series of patients subjected to subtotal gastrectomy for duodenal ulcer. In 79 patients operated on between July 1, 1937, and June 30, 1940, there was an operative mortality of 10 per cent, whereas in 94 patients operated on during the following three-year period, the mortality was 4 per cent. The authors believe that better management of the duodenal stump with preliminary and complementary jejunostomy accounted for the improvement in results.

Rienhoff[32] reports his results in 260 patients operated on for duodenal ulcer and followed for two to ten years. Two hundred and four patients (78 per cent) were considered well. Of the latter, complications developed in 32 (15 per cent), 22 of whom were subjected to further surgery. Thirty patients (12 per cent) were considered improved, and 4 of these were reoperated on. Twenty-one patients (8 per cent) were classified as unimproved, and 3 of these were reoperated on. The percentage of patients who remained well was higher in those with lowered gastric acidity than in those whose acidity continued high. Twenty-three patients (9 per cent) of the entire series developed anastomotic or jejunal ulcers. Rienhoff states that half the distal stomach with the pylorus was removed in all the patients, a short-loop anticolic anastomosis being used. In over 75 per cent of the cases, the ulcer was not resected. The operative mortality in the first 100 cases was 5 per cent. There were no deaths in the last 160 patients operated on, but some of these operations were too recent for end-result studies.

Lahey[33] advises in some duodenal ulcer operations the use of a T tube in the common bile duct with a long arm through the papilla of Vater for ease of identification in difficult dissections. In his discussion of Rienhoff's paper, he brings out the likelihood of better results if the ulcer can be safely included in the resection. Marshall[34] believes that a more radical resection of the stomach than Rienhoff uses would result in fewer jejunal ulcers.

McKittrick, Moore and Warren[35] analyzed the gastric resections for ulcer in the Massachusetts General Hospital from 1936 to 1943. There were 2 deaths in 106 cases of gastric ulcer and 10 deaths in 218 cases of duodenal ulcer — an over-all operative mortality of 3.7 per cent. These authors stress the dangers that arise in management of the duodenal stump in duodenal ulcer and recommend more frequent use of a two-stage procedure. The Finsterer subtotal gastrectomy for exclusion is done as the first stage, and the removal of the antrum and the first portion of the duodenum as the second stage six weeks later. St. John[36] in his discussion of this paper reports the results in the Presbyterian Hospital, New York City between 1939 and 1943. The gastric and duodenal ulcer operations are grouped together, with an over-all mortality rate of 3.7 per cent in 299 cases.

Lannin[37] gives an excellent review of the development of the modern concept of adequate resection of the stomach for duodenal ulcer. Experimental work based on duodenal ulcers in dogs produced by the method of Code and Varco[38] showed that the only procedures that always cured these ulcers were gastrectomy with sacrifice of the distal two thirds of the stomach and the Finsterer exclusion operation with removal of the antral mucosa. These writers further show by their experiments that the anastomosis must be placed as close as possible to the ligament of Treitz. They give evidence that antecolic anastomosis results in jejunal ulcer in direct proportion to the length of the afferent loop. This is supported by a series of three hundred subtotal gastrectomies in patients with duodenal ulcer. With these features in mind, no anastomotic or jejunal ulcer has so far developed. They believe that this is due to three reasons: the distal two thirds of the stomach is sacrificed; no antral mucosa is left behind; and the anastomosis is made as near the duodenojejunal junction as is technically possible.

Dragstedt and Schafer[39] give a further report on the treatment of peptic ulcer by transthoracic vagus interruption. Thirteen patients with duodenal ulcer, 1 with gastric ulcer and 1 with gastrojejunal ulcer were treated by this method. Three of the patients with duodenal ulcer were later subjected to gastroenterostomy for persistent duodenal obstruction. All these patients were immediately relieved of their ulcer symptoms, since hypersecretion of gastric juice was eliminated, but 1 patient had a recurrence of symptoms after three months of relief. The patient with gastrojejunal ulcer required a prolonged medical regimen after vagus interruption and was only partially relieved of his symptoms.

Moore, Chapman, Schulz and Jones,[40] have been studying this attack on the ulcer problem at the Massachusetts General Hospital. Their preliminary report substantiates Dragstedt's claims. These patients are immediately relieved of ulcer pain. The procedure is particularly adaptable to the recurrent-ulcer group and may well supplant subtotal gastrectomy in cases of uncomplicated but intractable ulcer. The wisdom of using this operation for cicatricial stenosis of the duodenum is doubtful. It probably has a limited field in gastric ulcer owing to the high incidence of early cancer in these patients.

Small Intestine

Cleland[41] measured the length of the small intestine in 100 cases at autopsy. It was impossible to determine that age, sex or disease had any bearing on the variations, which ranged from 4 to 11 meters. The physiologic processes of the subjects examined seemed undisturbed.

These data are particularly important to the clinician. Reports are often made on the successful removal of considerable lengths of the small bowel with apparent subsequent good health. On the other hand, patients may suffer a serious handicap from the loss of a relatively small segment of bowel. Occasionally this produces problems concerning nutrition, but the disturbance oftenest seen is one of persistent diarrhea. It is quite possible that the original length of the intestine in a given patient needing elimination of a segment of bowel may influence the postoperative course. A rapid intestinal rate of a functional type also plays a role in this problem.

Smith[42] has analyzed the experience in the Presbyterian Hospital in 1000 cases of intubation with the Miller–Abbott tube, which was successfully used in 751 cases and failed in 219, with the records incomplete in 30. It was employed prophylactically in 151 lesions of the small bowel and postoperatively in 84, and prophylactically in 108 lesions of the large bowel and postoperatively in 15; 153 intubations were for diagnostic purposes. Two or three cubic centimeters of mercury in the balloon is advised to aid the passage of the tube through the pylorus. There were complications in 140 cases—trivial in 129 and serious in 11. The tube required replacement in 54 cases.

Deering,[43] in reporting on his experience with the double-lumen tube in 200 cases, points out that the effectiveness of Wangensteen suction on a single-lumen nasal tube must not be forgotten. He believes that the Miller–Abbott tube should be reserved for extensive distention of the small bowel, the simpler method being more adaptable to the prevention of postoperative ileus and the relief of high intestinal obstruction.

Garlock and Crohn[44] analyzed the results in 164 patients treated for regional ileitis and ileocolitis. In 65 patients, the ileum was transected proximally to the disease and the distal end was inverted and dropped free into the peritoneal cavity, this being followed by ileotransverse colostomy. There were no deaths following this procedure, and recurrence of symptoms required extirpation of the diseased segment in only 9 cases. One-stage resection was done in 55 patients, of whom 9 died and 9 had recurrences. Twenty-five patients had two-stage resections, with death in 3 cases and a recurrence in 8. In 19 cases of ileocolitis, there were 2 deaths and 3 cases with recurrence.

In 19 patients who were subjected to resection for regional ileitis by Bockus,[45] 3 died and 9 developed recurrent disease.

Cave[46] had 3 deaths, all from perforation of the ileum, in a series of 23 patients with regional ileitis. One of these died preoperatively, 1 a year after operation and 1 two years after operation. Seven of 80 patients operated on for ulcerative colitis had involvement of the terminal ileum. Cave calls attention to a rare form of chronic inflammation that he has encountered once, with a successful outcome. In this type there is a fibrinous envelope surrounding the intestine, which causes obstruction by infolding of the mucous membrane from outside pressure. Relief was obtained by the removal of the encapsulating structure.

Two unusual double intussusceptions of the ileum have been reported.[47, 48] Both cases were in boys, aged four and five years. The first patient following treatment for round worms developed intussusception of the ileum with a second loop extending into the original one. The second case was initiated by a lipoma in the wall of the ileum. The first patient recovered after reduction of the intussuscepted segments, and the second after resection and anastomosis.

Dearing et al.[49] present an interesting case history of a seventy-five-year-old man who was admitted with intermittent intestinal obstruction and weight loss. Study revealed a calculus in the ileum, which was successfully removed at operation. It weighed 70 gm., measured 5.5 by 5.5 cm., and consisted chiefly of calcium carbonate and calcium phosphate.

Stone[50] presents the results of studies on patients with massive melena. Of 72 patients varying in age from eleven months to seventy-nine years, 21 had a cause for the bleeding proved by examination, operation or autopsy; in 20 cases, a possible but not a definite cause was revealed; in 31 patients, no cause for melena could be found. Stone points out that the latter group of cases should be treated conservatively and future studies done. Exploratory laparotomy, he states, may be justified in a search for the cause in certain patients at a time of election.

In a study of cancer in the small intestine, Shallow, Eger and Carty[51] brought out the fact that in routine autopsies such lesions are found in the colon thirty-six times as often as in the small bowel. To 269 cases found in the literature they added 38 cases from the records of the Jefferson Hospital, Philadelphia. Carcinoma was most frequent in the duodenum and sarcoma most frequent in the ileum. The operative mortality, which was 44 per cent, was highest in the lesions of the duodenum and lowest in those of the jejunum.

To the above cases may be added 21 cases of malignant tumors of the small intestine studied by Fraser[52] in Glasgow. In his report, lesions of the duodenum were excluded and the ratio of carcinoma to sarcoma was 12:9.

Hanno and Mensh[53] report a case of intermittent massive melena of fourteen years' duration. The patient had been subjected to gastroenterostomy on an erroneous diagnosis. Bouts of hemorrhage continued and finally ended in death from acute anemia. Autopsy revealed as the cause of bleeding a leiomyoma of the jejunum 20 cm. distal to the gastroenterostomy stoma.

Sibley[54] reviews the literature on Meckel's diverticulum and reports 10 cases from his own experience. Emphasis is placed on the high incidence of gastric mucosa found in these lesions and on the frequency of dyspepsia resulting from hypersecretion at this level in the gastrointestinal tract. He emphasizes that the possibility of this condition as a cause of abdominal pain is frequently overlooked. Gendel and Beaver[55] have successfully treated a patient with Meckel's diverticulum still attached to the umbilicus. This case was complicated by gastric mucosa and by a wood splinter in the diverticulum that caused intestinal obstruction. An incidental finding of early acute appendicitis seemed to play no recognizable role in the symptomatology. An additional case of Meckel's diverticulum containing calculi has been reported by Allen and Donaldson,[56] making a total of 11 recorded cases.

Colon

DeBakey and Carter[57] discuss penetrating wounds of the abdomen and bring out the fact that the mortality rate in World War I was 45 per cent, as compared with 15 to 20 per cent in World War II. They attribute these better results to three chief factors: ample supplies of whole blood and plasma; a larger number of well trained surgeons in the forward area; and the widespread use of exteriorization of colon wounds, routine proximal colostomy and proper drainage of the pelvis in wounds of the rectum. The authors lay too little emphasis on the effects of better selection and distribution of personnel by the Office of the Surgeon General.

Poth[58] has made an exhaustive report on collected data relating to the use of sulfasuxidine and sulfathalidine in surgery of the colon. The effect of these drugs in preoperative cleansing of the bowel has, in the opinion of many surgeons, proved to be a good adjunct to careful colonic surgery.

Newton and Blodgett[59] compare the results in two series of colon operations done at the Peter Bent Brigham Hospital. In 78 resections without the use of sulfasuxidine, there were complications in 45 and 15 deaths. In 36 patients treated preoperatively with sulfasuxidine, there were complications in 9 cases and 1 death following operation.

Bargen[60] has used both sulfasuxidine and sulfathalidine extensively in intestinal disease. He points out the advantage of the lower toxicity of sulfathalidine and reports that some patients sensitive to sulfasuxidine tolerate sulfathalidine well. He believes, as we do, that sulfathalidine is the superior drug. It gives a better preparation of the bowel and is less apt to cause bleeding from the growth and nausea to the patient.

Coller and Vaughan[61] report a series of 173 consecutive cases of carcinoma of the colon recently treated at the University of Michigan Hospital. Twenty-eight cases (16 per cent) were not resectable. Of the 145 cases considered operable, 28 (19 per cent) had gross metastases to the liver or peritoneum. There were 112 patients whose spread of disease appeared to be limited to the resectable nodal regions. In this group there was only 1 death, which followed an attempt to remove widespread disease. These authors preferred the two-stage attack on the right colon if the lesion was large or infected or produced obstruction and if the patient was debilitated. One-stage procedures were done on more favorable lesions of the right colon and in all other segments. Fourteen patients had the Rankin obstructive modification of the Mikulicz operation. All the others had primary anastomosis, which in 68 cases was of the aseptic and in 52 was of the open type. Delayed wound closure was employed when there was gross contamination or after the open method of anastomosis. Sulfonamides were not used in the peritoneal cavity. Transverse incisions with fine-wire closure and early ambulation are believed to have contributed to the good results.

White and Amendola[62] report the results of colon resection for malignant disease in the Roosevelt Hospital, New York City. It appears that primary aseptic suture and proximal decompression resulted in half the deaths that occurred in modifications of the Mikulicz type of resection. These writers stress the shorter period of morbidity and the greater comfort to the patient in the primary anastomosis group.

Berson and Berger[63] found the incidence of multiple carcinomas of the colon to be 5 per cent of all their colonic lesions. Thirteen patients had two areas of carcinoma at the same time, and 3 had three areas. Polyps were present in 25 per cent of the patients having one area of carcinoma and in 38 per cent of those with multiple lesions. Sixty-seven per cent of the polyps in the multiple group had undergone malignant degeneration. These authors point out the need for thorough investigation of the entire colon and the importance of follow-up studies.

Pregnancy complicating carcinoma of the colon is discussed by Banner, Hunt and Dixon.[64] They found 62 cases in the literature and added 7 from the Mayo Clinic. They point out that the usual symptoms of carcinoma of the colon, such as nausea and constipation, are often associated with pregnancy and are therefore overlooked. They are impressed with the ease of delivery after abdominoperineal resection and the apparent safety of

cesarean section in the presence of a colostomy.

Finn and Lord[65] report a case of carcinoma of the sigmoid with obstruction occurring in a thirty-one-year-old tripara who was in the sixth month of pregnancy. The lesion was treated by a three-stage procedure and the patient was successfully delivered at term.

Wangensteen[66] has anastomosed the colon to the rectal ampulla after resection in 24 patients for carcinoma of the rectum and rectosigmoid and in 3 patients for ulcerative colitis. There were 2 hospital deaths in the cancer group. He drains the pelvis posteriorly and expects a temporary fistula. The sphincter control was good in all cases. Three of the patients with cancer had local metastases to the liver, which were excised. Five of the 22 survivors had developed local pelvic recurrences at the time of the report. Wangensteen admits that fixed tumors require abdominoperineal resection.

Bacon[67] states that in 208 cases in which resection of the rectum was done for carcinoma he preserved the anal sphincter in 80 per cent. He has modified the combined abdominoperineal operation of Babcock to include preservation of the sphincter, and believes that this operation does not influence local recurrence provided that the muscles are not involved. He agrees with Babcock that perineal colostomy is superior to the abdominal type. His resectability rate was 81 per cent, and the mortality 5.5 per cent.

The increasing tendency to preserve the anal sphincter in carcinoma of the rectosigmoid and high rectum should be based on sound cancer surgery. One may make a low anastomosis and do Bacon's operation, or even extirpate the rectum through the perineal route with revision of the sphincter, under two conditions — in patients with early low-grade localized lesions at a suitable level and in those with an incurable spread of disease. Local recurrence is justifiable only in the latter group. The use of drainage to the pelvis incites a fistula, increases infection and produces subsequent stenosis of the suture line. If an adequate preparation is done before operation and the peritoneal reconstruction is made with room for adequate seepage of serum into the peritoneal cavity, the results without drainage are better.

Griffin, Barton and Meyer[68] found that volvulus of the sigmoid accounted for 8 per cent of the obstructions of the large bowel in a series of 458 cases admitted to the Cook County Hospital. Twenty-five records were analyzed in detail. There seemed to be two distinct types of sigmoid volvulus. There were 7 acute cases, mainly in young patients with an average of twenty-four hours of symptoms and no previous history of bowel disturbance. Eighteen cases were classified as subacute and were in the older age group. These patients were ill for an average of one hundred and two hours before admission, and many of them had had previous attacks. These authors call attention to a characteristic so-called "ace of spades" appearance in the roentgen-ray film after barium enema. The mortality in this series was 40 per cent. The authors stress early diagnosis and early resection and advise against conservative measures.

Pancreas

Morton[69] has given an excellent discussion on acute pancreatitis. Two thirds of the cases are those of acute pancreatic edema, and 60 per cent of these are associated with lesions in the biliary tract. The serum amylase is always elevated early in this type of the disease. Conservative treatment is indicated, and the mortality is low. Recurrent attacks are frequent. After recovery from the acute phase the biliary system should be studied, and if gallstones are present, they should be removed. Pancreatic necrosis accounts for the other third of patients with acute pancreatic disturbance. In this type of case the serum amylase may not be elevated and the symptoms are severer. Morton believes that surgery should be undertaken to establish adequate drainage of the pancreatic area as early in the disease as is compatible with the patient's condition. All nine patients with this necrosing type of pancreatitis who were treated conservatively died, whereas of those operated on for drainage 45 per cent recovered. Diabetes followed in a certain number of the recoveries. This author calls attention to the precipitation of acute pancreatic edema by operation for duodenal ulcer penetrating into the pancreas.

Shallow et al.[70] report a successful outcome in an extremely ill woman of fifty-five who had pancreatic necrosis of eight days' duration. In addition to a large pancreatic abscess, there were two abscess cavities in the quadrate lobe of the liver. They believe that penicillin and sulfadiazine aided in the recovery on the basis of the types of organisms cultured.

Ten patients with pancreatic collections operated on with recovery are reported by Pinkham.[71] He discusses the differential diagnosis between so-called "true cysts," on the basis of proliferative changes due to neoplasm, hydatid disease, congenital defects and retention due to obstruction of pancreatic ducts, and "pseudocysts." The latter type should be referred to simply as collections secondary to pancreatic trauma, inflammation or necrosis.

Whipple[72] records his results in pancreaticoduodenectomy. He believes that the one-stage procedure has many advantages over his original two-stage operation. Twenty-two patients with cancer and 5 with calcification of the pancreas were subjected to radical operation. In 8 cases in which the two-stage operation was done there were 3 deaths, whereas in 19 one-stage procedures there were 6 deaths. A patient who had islet-cell carcinoma was apparently well five years after opera-

tion. Whipple advises implanting the common duct in the jejunum, with implantation of the pancreatic duct slightly lower down. This avoids the possibility of bile leakage and eliminates the debated question concerning the importance of re-establishing pancreatic continuity with the gastrointestinal tract.

Orr and Walker[73] stress the relative infrequency of carcinoma of the duodenum and report a case of a successful Whipple procedure in one stage. They point out that most of the carcinomas in this region start in the ampulla of Vater and are improperly recorded as duodenal lesions.

Strode[74] reports a successful Whipple operation for carcinoma that was primary in a duodenal diverticulum.

Brunschwig et al.[75] record a case of total pancreatectomy, total gastrectomy, total duodenectomy, splenectomy, left adrenalectomy and omentectomy in a diabetic patient fifty-three years of age. The patient required no increase in the amount of insulin used preoperatively. He died of widespread carcinomatosis three and a half months after operation.

266 Beacon Street

References

1. Lemmon, W. T., and Hager, H. G., Jr. Continuous spinal anesthesia: observation on 2000 cases. *Ann. Surg.* **120**:129-142, 1944.
2. Arrowood, J. G., and Foldes, F. F. Subarachnoid analgesia maintained by continuous drop method. *Arch. Surg.* **49**:241-244, 1944.
3. Singleton, A. O., and Stehouwer, O. W. Fascia-patch transplant in repair of hernia. *Surg., Gynec. & Obst.* **80**:243-251, 1945.
4. Mair, G. B. Preliminary report on use of whole skin-grafts as substitute for fascial sutures in treatment of herniae. *Brit. J. Surg.* **32**: 381-385, 1945.
5. Skinner, H. L., and Duncan, R. D. Recurrent inguinal hernia. *Ann. Surg.* **122**:68-84, 1945.
6. Ochsner, A., and Johnston, J. H. Appendical peritonitis. *Surgery* **17**: 873-892, 1945.
7. McPherson, A. G., and Kinmonth, J. B. Acute appendicitis and appendix mass. *Brit. J. Surg.* **32**:365-370, 1945.
8. Rogers, H., and Faxon, H. H. Statistical study of six hundred and seventy-one cases of appendiceal-peritonitis. *New Eng. J. Med.* **226**:707-717 and 745-753, 1942.
9. Sewell, R. L. Appendicitis and sulfonamide drugs. *Surgery* **17**:32-38, 1945.
10. Bowen, W. H. Obstructive appendicitis. *Brit. J. Surg.* **32**:468-471, 1945.
11. Wilkie, D. P. D. Acute appendicitis and acute appendicular obstruction. *Brit. M. J.* **2**:959-962, 1914.
12. Knox, G. Lymphosarcoma primary in appendix: study of twenty-five cases. *Arch. Surg.* **50**:288-292, 1945.
13. Dixon, C. F., and Lichtman, A. L. Congenital absence of gall bladder. *Surgery* **17**:11-21, 1945.
14. Greenwald, W. Papilloma of gall bladder. *Surgery* **16**:370-376, 1944.
15. Smith, B. C. Acute cholecystitis. *S. Clin. North America* **25**:285-300, 1945.
16. Wallace, R. H., and Allen, A. W. Acute cholecystitis. *Arch. Surg.* **43**: 762-772, 1941.
17. Simon, M. M. Pitfalls to be avoided in cholecystectomy. *Am. J. Surg.* **66**:367, 1944.
18. Colp, R. Hepaticoduodenal intubation with hepatoduodenostomy for traumatic stricture of hepatic duct. *Surg., Gynec. & Obst.* **80**: 190-196, 1945.
19. Peterson, L. W., and Cole, W. H. Chronic sclerosing pancreatitis causing complete stenosis of common bile duct. *Arch. Surg.* **51**: 15-21, 1945.
20. Allen, A. W. Method of re-establishing continuity between bile ducts and gastro-intestinal tract. *Ann. Surg.* **121**:412-424, 1945.
21. Lahey, F. H. Strictures of common and hepatic bile duct. *Surg., Gynec. & Obst.* **80**:555, 1945.
22. Castle, W. B. Observations on etiologic relationship of achylia to pernicious anemia. *Am. J. M. Sc.* **178**:748-764, 1929.
23. Olson, S. W., and Heck, F. J. Lesions of gastric mucosa in pernicious anemia. *Proc. Staff Meet., Mayo Clin.* **20**:74-78, 1945.
24. Biggard, J. D., and Overmiller, W. Emergency gastrectomy for acute perforation of carcinoma of stomach with diffuse soiling of free peritoneal cavity. *Ann. Surg.* **120**:526-530, 1944.
25. Eggel, G. C. Creation of gastric pouch following total gastrectomy. *Surgery* **17**:512-523, 1945.
26. Marshall, S. F. Plan for surgical management of gastrojejunocolic fistula. *Ann. Surg.* **121**:620-638, 1945.
27. Ransom, H. K. Gastrojejunocolic fistula. *Surgery* **18**:177-190, 1945.
28. Graham, R. R., and Tovee, E. B. Treatment of perforated duodenal ulcers. *Surgery* **17**:704-712, 1945.
29. Price, P. B., and Lee, T. F. Use of omentum to close perforations of stomach. *Arch. Surg.* **50**:171-173, 1945.
30. Gray, H. K., and Sharpe, W. S. Problem of massive hemorrhage from duodenal ulcers of patients beyond middle life. *Ann. Surg.* **121**: 840-846, 1945.
31. Colp, R., and Klingenstein, P., Mage, S., and Druckerman, L. J. Subtotal gastrectomy for duodenal ulcer. *Ann. Surg.* **120**:170-180, 1944.
32. Rienhoff, W. F., Jr. Analysis of results of surgical treatment of 260 consecutive cases of chronic peptic ulcer of duodenum. *Ann. Surg.* **121**:583-599, 1945.
33. Lahey, F. H. Use of identifying "T" tube in common bile duct in gastric resection for duodenal ulcer adherent to bile ducts. *Surg., Gynec. & Obst.* **80**:197, 1945.
34. Marshall, S. F. Discussion of Rienhoff.[32]
35. McKittrick, L. S., Moore, F. D., and Warren, R. Complications and mortality in subtotal gastrectomy for duodenal ulcer: report on two-stage procedure. *Ann. Surg.* **120**:531-561, 1944.
36. St. John, F. B. Discussion of McKittrick, Moore, and Warren.[35]
37. Lannin, B. G. Experimental evaluation of satisfactory operation for ulcer. *Surgery* **17**:712-742, 1945.
38. Code, C. F., and Varco, R. L. Chronic histamine action. *Proc. Soc. Exper. Biol. & Med.* **44**:475-477, 1940.
39. Dragstedt, L. R., and Schafer, P. W. Removal of vagus innervation of stomach in gastroduodenal ulcer. *Surgery* **17**:742-749, 1945.
40. Moore, F. D., Chapman, W. P., Schulz, M. D., and Jones, C. M. Transdiaphragmatic resection of the vagus nerves for peptic ulcer. *New Eng. J. Med.* **234**:241-251, 1946.
41. Cleland, J. B. Length of small intestine. *M. J. Australia* **2**:359, 1944.
42. Smith, B. C. Experiences with Miller-Abbott tube. *Ann. Surg.* **122**:253-259, 1945.
43. Deering, W. H. Double lumen balloon-type tube as adjunct in management of intestinal obstruction. *Proc. Staff Meet., Mayo Clin.* **19**:600-602, 1944.
44. Garlock, J. H., and Crohn, B. B. Appraisal of results of surgery in treatment of regional ileitis. *J. A. M. A.* **127**-205-208, 1945.
45. Bockus, H. L. Present status of chronic regional or cicatrizing enteritis. *J. A. M. A.* **127**:449-456, 1945.
46. Cave, H. W. Inflammatory lesions of small intestine. *J. A. M. A.* **127**:456-458, 1945.
47. Milroy, P. Unusual ileoileal intussusception. *Arch. Surg.* **50**:307, 1945.
48. Case records of Massachusetts General Hospital (Case 31041), *New Eng. J. Med.* **232**:113-115, 1945.
49. Dearing, W. H., Berkman, J. M., Waugh, J. M., and Pugh, D. G. Partial intermittent intestinal obstruction caused by enterolith in ileum. *Proc. Staff Meet., Mayo Clin.* **19**:436-438, 1944.
50. Stone, H. B. Large melana of obscure origin. *Ann. Surg.* **120**:582-597, 1944.
51. Shallow, T. A., Eger, S. A., and Carty, J. B. Primary malignant disease of small intestine. *Am. J. Surg.* **69**:372-383, 1945.
52. Fraser, K. Malignant tumors of small intestine: review of literature and report of 21 cases. *Brit. J. Surg.* **32**:479-491, 1945.
53. Hanno, H. A., and Mensh, M. Leiomyoma of jejunum: intermittent melena of 14 years' duration and fatal hemorrhage. *Ann. Surg.* **120**: 199-206, 1944.
54. Sibley, W. L. Meckel's diverticulum: dyspepsia Meckeli from heterotopic gastric mucosa. *Arch. Surg.* **49**:156-166, 1945.
55. Gendel, S., and Beaver, M. G. Unusual case of Meckel's diverticulum. *Ann. Surg.* **121**:891-893, 1945.
56. Allen, A. W., and Donaldson, G. A. Meckel's diverticulum containing calculi. *Arch. Surg.* **50**:286, 1945.
57. DeBakey, M. E., and Carter, B. N. Current considerations of war surgery. *Ann. Surg.* **121**:545-562, 1945.
58. Poth, E. J. Succinylsulfathiazole and phthalylsulfathiazole in surgery of colon. *Surgery* **17**:773-780, 1945.
59. Newton, F. C., and Blodgett, J. B. Succinylsulfathiazole and intestinal suction in surgery of large bowel. *Surgery* **18**:200-206, 1945.
60. Bargen, J. A. Sulfathalidine in intestinal disease. *Proc. Staff Meet., Mayo Clin.* **20**:85-87, 1945.
61. Coller, F. A., and Vaughan, H. H. Treatment of carcinoma of colon. *Ann. Surg.* **121**:395-411, 1945.
62. White, W. C., and Amendola, F. H. Advantages and disadvantages of closed resection of colon. *Ann. Surg.* **120**:572-581, 1944.
63. Berson, H. L., and Berger, L. Multiple carcinomas of large intestine. *Surg., Gynec. & Obst.* **80**:75-83, 1945.
64. Banner, E. A., Hunt, A. B., and Dixon, C. F. Pregnancy associated with carcinoma of large intestine. *Surg., Gynec. & Obst.* **80**:211-216, 1945.
65. Finn, W. F., and Lord, J. W., Jr. Carcinoma of colon producing acute intestinal obstruction during pregnancy. *Surg., Gynec. & Obst.* **80**: 545-548, 1945.
66. Wangensteen, O. H. Primary resection (closed anastomosis) of rectal ampulla for malignancy with preservation of sphincteric function. *Surg., Gynec. & Obst.* **81**:1-24, 1945.
67. Bacon, H. E. Evolution of sphincter muscle preservation and re-establishment of continuity in operative treatment of rectal and sigmoidal cancer. *Surg., Gynec. & Obst.* **81**:113-127, 1945.
68. Griffin, W. D., Barton, G. R., and Meyer, K. A. Volvulus of sigmoid colon. *Surg., Gynec. & Obst.* **81**:287-294, 1945.
69. Morton, J. Acute pancreatitis. *Surgery* **17**:475-491, 1945.
70. Shallow, T. A., Eger, S. A., and Wagner, F. B., Jr. Suppurative pancreatitis with associated liver abscess. *Ann. Surg.* **121**:853-859, 1945.
71. Pinkham, R. D. Pancreatic collections (pseudocysts) following pancreatitis and pancreatic necrosis: review and analysis of 10 cases. *Surg., Gynec. & Obst.* **80**:225-235, 1945.
72. Whipple, A. O. Pancreaticoduodenectomy for islet carcinoma. *Ann. Surg.* **121**:847-852, 1945.
73. Orr, T. G., and Walker, G. A. Pancreaticoduodenectomy for primary carcinoma of cuodenum. *Surg., Gynec. & Obst.* **80**:149-151, 1945.
74. Strode, J. E. Radical duodenopancreatectomy: report of successful resection of a carcinoma of duodenal diverticulum involving head of pancreas. *Surgery* **18**:115-129, 1945.
75. Brunschwig, A., Ricketts, H. T., and Bigelow, R. R. Total pancreatectomy, total gastrectomy, total duodenectomy, splenectomy, left adrenalectomy and omentectomy in diabetic patient: recovery. *Surg., Gynec. & Obst.* **80**:252-256, 1945.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 32121

PRESENTATION OF CASE

A twenty-three-year-old woman was admitted to the hospital complaining of vaginal bleeding.

She was in the fourth month of pregnancy. Ten days before admission the fetus was said to have died, and six days later vaginal bleeding began and increased up to admission.

The past history revealed that the patient had been in good health until a year before admission. At that time she had a spontaneous abortion at about the third month of pregnancy. She had received a plasma transfusion.

On physical examination the patient was well developed and well nourished and in no acute discomfort. The heart and lungs were normal. The uterus showed enlargement consistent with a four months' pregnancy. There was a foul, blood-stained vaginal discharge.

The temperature was 101°F., the pulse 80, and the respirations 20. The blood pressure was 110 systolic, 50 diastolic.

The red-cell count was 4,240,000, with 12.2 gm. of hemoglobin. The white-cell count was 21,400, with 86 per cent neutrophils; on smear, many band forms were seen and the platelets were decreased. On the first hospital day the sediment from a catheterized specimen of urine contained 25 red cells, an occasional white cell and 5 epithelial cells per high-power field.

Soon after admission the patient was sedated with codein and aspirin and given 6 gm. of sulfadiazine. Penicillin was ordered, 16,000 units every two hours. Later the dose was increased to 64,000 units every two hours. During the afternoon of the first hospital day the temperature rose to 105°F. She had a chill and a few hours later spontaneously passed a macerated fetus. Fifteen minutes afterward an ampule of Pituitrin was administered intravenously, and a half ampule at fifteen-minute intervals for two more doses. The placenta, which was yellow, friable and necrotic, was passed at the time of the second dose. Eleven milligrams of morphine were given. After the abortion the patient complained of severe chest pain. The blood pressure fell steadily to a level of 40 systolic, 0 diastolic. An intravenous injection of dextrose in saline solution was begun, and 1 unit of plasma was given. The patient's condition improved somewhat, and the intravenous drip was continued. Four hours later she was found in shock, being only slightly responsive, with a blood pressure of 40 systolic, 0 diastolic, and a pulse of 120 to 150. Another unit of plasma was given, followed by a transfusion of whole blood. A few hours later another whole-blood transfusion was given.

During the second hospital day, the patient became more responsive. She complained of nausea and severe generalized aching. Despite continuous intravenous drip and hypodermoclysis, she remained dehydrated. The blood pressure rose to about 50 systolic, with a pulse of 145 that was of better quality. She remained oliguric, only 30 cc. of urine being obtained by catheter during twenty-four hours. The urine was cloudy, light amber and acid and gave a + test for albumin. The quantity was insufficient for measurement of the specific gravity. There was no sugar or diacetic acid. The sediment contained a rare coarsely granular cast, a rare red cell, an occasional white cell, 5 round cells and 10 epithelial cells per high-power field. No crystals were seen. The urine was sterile. A marked generalized flush appeared, with definite circumoral pallor. She vomited about 150 cc. of grayish material mixed with mucus. She manifested air hunger, the respirations being deep and sighing. She was not cyanotic. The lungs were clear. She was given a transfusion of Rh— blood, 3500 cc. of fluid, 7 gm. of sodium bicarbonate and 100 cc. of cortical extract.

On the third hospital day, periorbital edema appeared but the lungs remained clear. The blood pressure rose to 80 systolic, 50 diastolic. The generalized erythema became more punctate. Mild air hunger persisted. There was marked epigastric tympany, with fullness. Peristalsis was sluggish but of normal pitch. A few drops of urine were obtained by catheter. The sediment contained many white cells and a few red cells per high-power field; there were no casts or crystals. The patient was alert. Herpes labialis appeared. Cystoscopy revealed a normal bladder containing no urine. Both ureters were easily catheterized but no urine was obtained. The ureteral catheters were left in place.

On the fourth hospital day the serum protein was 3.5 gm. per 100 cc. A Schultz–Charlton test was negative. The patient became semicomatose, with fibrillary twitching of the entire body. A subconjunctival hemorrhage appeared in the right eye. Early on the fifth hospital day she had a severe generalized convulsion and expired.

DIFFERENTIAL DIAGNOSIS

DR. ARTHUR T. HERTIG:* This case may be summarized as one of a young woman who had had no

*Pathologist, Boston Lying-in Hospital and Free Hospital for Women, Brookline.

previous full-term pregnancies but had had one previous miscarriage of spontaneous origin and who came in with a septic inevitable abortion in the fourth month of pregnancy. The first thing of interest is the fact that this spontaneous inevitable abortion was septic. That in itself is an extremely unusual fact. Very seldom do spontaneous abortions show a septic course. I assume that this was a spontaneous rather than an induced abortion and that she was febrile from it rather than from some other inflammatory process.

Another interesting factor is that the patient bled so soon after the fetus died. The latter event occurred about ten days before delivery, and she began to bleed six days later, four days prior to admission. Usually it takes longer after fetal death for the sequence of events resulting in abortion to take place. The most immediately interesting thing about this patient is that she delivered spontaneously and then required two ampules (2 cc.) of intramuscular Pituitrin in divided doses to effect delivery of the placenta. Pituitrin was indicated since it is good obstetric procedure not to invade the uterus in a case of septic abortion because of the chance of spreading the infection.

The first thing of importance with respect to her death seems to me to be the fact that she reacted badly, almost immediately, to the administration of Pituitrin. It must be remembered too that there could have been other causes for the patient's going into shock, one being unrecognizable or undiagnosed rupture of a viscus, specifically a ruptured uterus. I take it that the clinicians involved were satisfied that the patient had no ruptured internal viscus. She certainly had no external bleeding or peritoneal signs to go with a ruptured uterus. Hence, I should say that in all probability she did not have a ruptured uterus, although that must be considered. My impression at this point is that the patient had profound Pituitrin shock and was one of the very rare persons sensitive to even moderate doses of this drug.

The next fact of importance is that prior to delivery a catheter specimen of urine contained 25 red cells per high-power field. Whether the red cells came as a result of the trauma of catheterization or whether she actually had a hematuria, I do not know. That is an important point to consider in view of the oliguria and anuria that developed later. A significant fact clinically is that she was in general shock from which she did not respond well despite intravenous fluid, plasma transfusion and two whole-blood transfusions. It is of interest to ask why she did not respond to an apparently adequate amount of fluid and blood in the absence of a ruptured viscus and in the absence of any profound loss of blood. These features help to make the diagnosis of Pituitrin shock.

The next point to inquire about is whether this patient was Rh—, and I assume that she was since they gave Rh— blood on the third transfusion. If she was Rh—, the next thing to determine is whether she had been previously sensitized by the Rh factor either from a previous transfusion or by the previous pregnancy, which aborted at three or four months. So far as I know there are no examples of isoimmunization in an Rh— patient by a pregnancy that did not persist over three or four months. She may, however, have been given Rh+ blood on the two transfusions before the Rh— transfusion. Is there any information about that?

DR. WADE VOLWILER: They were Rh+.

DR. HERTIG: Then that is a possible factor in her death, namely, that she had been sensitized to Rh+ blood, developed agglutinins and reacted fatally on perhaps the second of the two transfusions. Were both of them Rh+?

DR. VOLWILER: Yes.

DR. TRACY B. MALLORY: How long was the time interval between the first and the last transfusion?

DR. VOLWILER: Not more than three days.

DR. HERTIG: And the time interval between the first two was a matter of hours?

DR. VOLWILER: Yes.

DR. HERTIG: So on the basis of the history one can rule out reaction of the patient to Rh+ blood after sensitization. There had not been time for her to have become sensitized. Furthermore, she does not appear to have had an ordinary transfusion reaction.

The next point of clinical significance is the fact that she had oliguria, which progressed to almost complete anuria. In a pregnant patient who had previously been transfused that could suggest two things — transfusion reaction, which can largely be ruled out, and bilateral symmetrical cortical necrosis of the kidney. Renal cortical necrosis was probably brought on by severe prolonged ischemia, induced by Pituitrin sensitivity and resulting in contraction of the precapillary arterioles throughout the body, particularly the afferent and efferent arterioles of the kidney. Hence my diagnosis is bilateral symmetrical cortical necrosis of the kidney. The clinical course is typical. She died at about the average time, namely five days. I do not believe that she had a transfusion reaction, nor do I believe that she had a ruptured internal viscus. The profound, prolonged shock is difficult to evaluate.

DR. MALLORY: Are there any questions or other diagnoses that anyone would like to suggest?

A PHYSICIAN: Could syphilis be connected with this story in any way?

DR. MALLORY: I believe not. The Hinton test was negative.

DR. W. SCHIER WILSON: Were hematein crystals seen in the urine on any occasion?

DR. MALLORY: There is no record of them.

DR. HERTIG: In fact, it states that there were no crystals of any sort. I should have said that this case might possibly be one of sensitization or sen-

sitivity to a sulfonamide because of the fact that some persons are sensitive to such drugs and react to relatively small doses. The patient received 6 gm. of sulfadiazine. Such patients, — that is, the few whom I have seen, — die on the average in five or

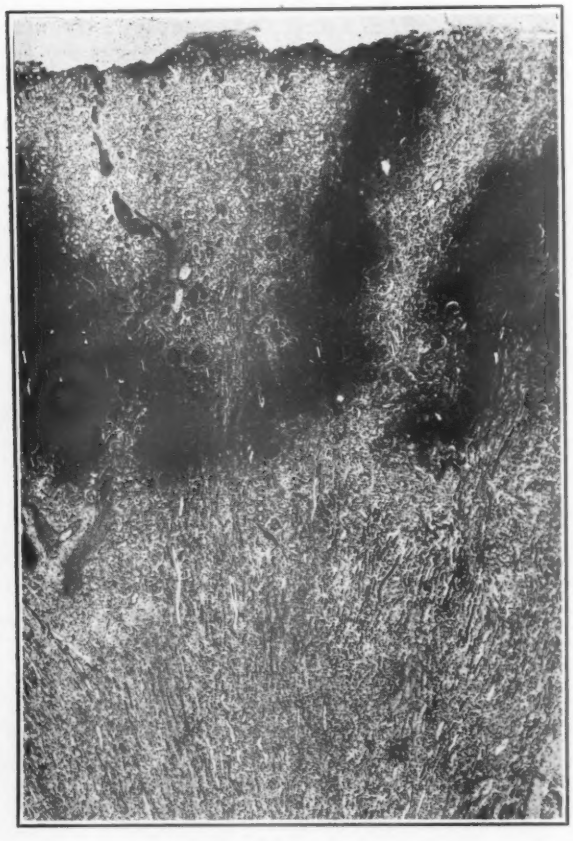

FIGURE 1.

six days with anuria. In my opinion, however, there should have been some evidence of sulfonamide crystals in the urine were this case one of sensitivity to one of these drugs.

CLINICAL DIAGNOSES

Spontaneous abortion with septicemia.
Uremia.

DR. HERTIG'S DIAGNOSIS

Bilateral cortical necrosis of kidneys.

ANATOMICAL DIAGNOSES

Symmetrical necrosis of renal cortex.
Infarction of adrenal gland, bilateral.
Subinvolution of uterus.
(Uremia.)

PATHOLOGICAL DISCUSSION

DR. MALLORY: Except for the difference of sex and the presence of pregnancy this case is quite reminiscent of scores of cases that many of us saw overseas during the last couple of years and in which the pathologic lesion is entirely different from that Dr. Hertig has suggested. Almost any patient with severe traumatic shock who has received multiple transfusions runs a considerable risk of dying with delayed anuria and a clinical picture that, so far as I can see, is indistinguishable from the late stages of this story.

The kidneys of this patient were slightly enlarged. The external surfaces were vaguely mottled, poorly defined purplish areas being separated by irregular yellow areas. The appearance of the cut surface was much more striking. The entire cortex was discolored. The major part of it was bright red, but there were scattered, irregular, yellow areas in the midst of the hemorrhage. Under low power the cortex showed dark areas representing hemorrhage (Fig. 1). These stopped abruptly at the junc-

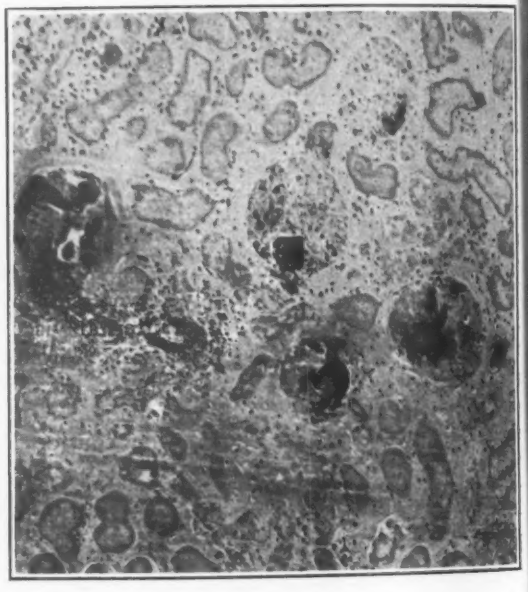

FIGURE 2.

tion between the cortex and pyramid. With higher power, the striking feature comprised the blood vessels. In a typical glomerulus and its afferent arteriole (Fig. 2), the wall of the arteriole appeared black; it was completely necrotic, part of it solidly thrombosed and part having a lumen. The fibrin of the thrombus extended over into several of the loops of the glomerulus. The tubules were widely separated by hemorrhagic stroma. The adrenal glands weighed 40 gm. and were completely necrotic, with hemorrhagic borders (Fig. 3). The picture essentially is like that of the Waterhouse–Friderichsen syndrome. The arterioles in the capsules of the

adrenal glands were necrotic (Fig. 4). The change is identical with that present in the kidneys.

Dr. Hertig's diagnosis of symmetrical cortical necrosis is substantiated, and as he pointed out, that is the usual lesion that one would expect in a patient who developed delayed uremia immediately after delivery. The lesion that we saw with shock in Europe was entirely different. There was no

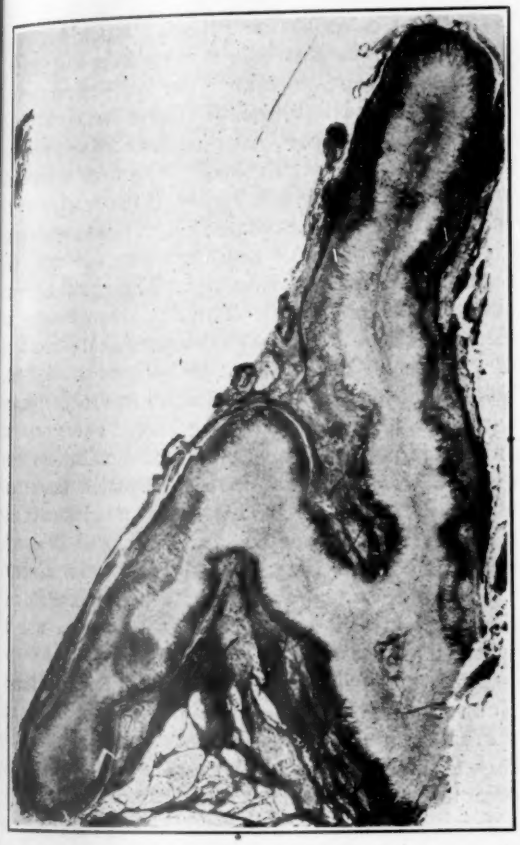

FIGURE 3.

necrosis of the cortex. The degenerative changes were found in the lower part of the nephron, the ascending loop of Henle in particular, and the tubules were filled with hemoglobin casts just as they are in a transfusion reaction.

Have you any further comment, Dr. Hertig?

DR. HERTIG: I have seen two cases like this. One patient died on the fifth day, and the other on the eleventh day post partum. The former case was exactly like the one presented here. The patient who lived eleven days did so only by the heroic efforts of the house staff, who did several exsanguination transfusions. The entire renal cortex of the latter case was necrotic, but instead of being mottled red and yellow, it had a brilliant orange-yellow border around the periphery of the kidney, with only minute areas of viable tissue left. It is our impression from studying these cases that the initial lesion was in the precapillary arterioles of the kidney. The precapillary arterioles elsewhere in the body, whether in the bowel, adrenal gland or pituitary gland or elsewhere, also showed involvement. The massive cortical necrosis was apparently due to retrograde or diffuse thrombosis of the small erect arteries, but the initial lesion began in the small precapillary arterioles. Is that your impression?

DR. MALLORY: I do not feel competent to express an opinion. This is a fairly rare condition. At the time Dr. Hertig* reported his cases four years ago he was able to collect about 70 cases from the literature. It is unquestionably not so rare as that suggests, because all of us know of unreported cases.

FIGURE 4.

It was first described in relation to pregnancy, and it is still seen much more frequently in relation to pregnancy than in any other condition. It can occur in the male sex, and a condition that simulates it closely can be produced in animals by injecting a toxin derived from staphylococci, which have nothing to do with the genesis of the human lesion.

DR. DONALD S. KING: How much of a factor was the Pituitrin?

*Sheldon, W. H., and Hertig, A. T. Bilateral cortical necrosis of kidney. *Arch. Path.* 34:866-874, 1942.

DR. MALLORY: I do not feel capable of answering that. Dr. Hertig has said all that can be said.

DR. JAMES B. TOWNSEND: Have all the patients received Pituitrin?

DR. HERTIG: I do not know whether they have or not. Most cases, however, follow so-called "toxic separation" of the placenta. In this case the clinical picture was so striking immediately following administration of Pituitrin that the drug seems to have had something to do with it. We know of course that this drug does contain a pressor substance affecting blood vessels and hence could have been the stimulus which caused the generalized arteriolar spasm that resulted, among other things, in cortical necrosis.

DR. TOWNSEND: Was any organism obtained from the blood ante or post mortem?

DR. MALLORY: No.

DR. HERTIG: It must be pointed out that most cases of this condition in pregnancy are associated with toxic separation of the placenta, the underlying pathology of which is an acute atheromatous degeneration of the small arterioles of the placental site. This causes decidual necrosis and separation of the placenta from hemorrhage.

DR. BENJAMIN CASTLEMAN: Was either of your cases as early in pregnancy as this?

DR. HERTIG: No; they were in the seventh and eighth months.

DR. MALLORY: Occasionally, as Dr. Hertig has pointed out, vascular lesions are found in other spots of the body besides the kidney. We were able to obtain the pituitary gland and cut a number of sections of it, but we did not find any vascular lesions.

CASE 32122

PRESENTATION OF CASE

A twenty-nine-year-old unmarried woman, a stenographer, was admitted to the hospital complaining of dizziness, nausea and vomiting. There is doubt concerning the validity of the history, since it was obtained from the patient, who was extremely ill.

She was well until about five weeks previous to admission, when she began to have intermittent attacks of weakness and dizziness. These apparently did not increase in severity or frequency. Five days previous to admission, while sleeping, she was suddenly awakened by severe vertigo, followed by severe nausea and vomiting. Immediately after this episode she felt weak. She again returned to sleep, but a few hours later she was awakened by the same symptoms, which lasted for about an hour. The following morning she had another similar attack. A local doctor was called, who thought that it was a gastrointestinal upset but later consulted a neurologist, who advised hospitalization. The patient stated that about twenty-four hours after th onset of her illness she developed a blurring of visio when looking through both eyes but not when look ing through one. During the twenty-four hour previous to admission the doctor had started slow digitalization by mouth, giving 0.1 gm. every fou hours. An undetermined amount of this wa vomited. The patient gave no history of rheumati fever, previous recent infection or the taking o drugs or medicine.

Physical examination revealed a pale woman i considerable distress who was retching and restless The pupils were normal. The fundi showed ques tionable binasal blurring. There was no hemi anopsia. The extraocular muscles were normal. Th neck was questionably stiff. The neck veins were not engorged. The left border of the heart was per cussed at the anterior axillary line. The sounds were distant. No thrills or murmurs were detected. The cardiac rhythm was irregular. The apical rate wa 108, the peripheral 65. The diaphragm was higher than usual. A few moist rales were heard over both lung bases. The liver edge was palpated 3 cm. below the right costal margin and was tender; otherwise the abdomen was negative. There was no ankle edema, and the dorsal-pedis pulsations were normal. Vaginal examination revealed tenderness of both lateral fornices; there were no masses or discharge. The reflexes were sluggish, and Babinski's sign was absent. There was no sensory impairment.

The temperature was 101.2°F., the pulse 56, and the respirations 20. The blood pressure was 92 systolic, 60 diastolic.

The white-cell count was 16,750, and the hemoglobin was 100 per cent (Sahli). The urine was normal. An electrocardiogram soon after admission showed auricular flutter, at a rate of 290. The QRS complexes were aberrant and slurred, occurring as a bigeminy at a rate of about 75. There was a question of 2:1 or 1:1 heart block alternating or of 4:1 heart block with ventricular premature beats as bigeminy.

A short time after admission the patient became flaccid, unconscious and cyanotic. The heart rate remained rapid and irregular. The blood pressure fell to 60 systolic, 40 diastolic. An electrocardiogram taken an hour after admission was similar to the first one, with occasional ventricular premature beats interrupting the rhythm. These originated from different foci in Lead 3. Special auricular Lead 1 showed short strips that appeared to confirm the diagnosis of auricular flutter. CF_1 and CF_2 showed upward deflections of the R waves.

Half an hour later 6 cc. of Cedilanid was given intravenously. The patient remained unconscious, but the blood pressure rose to 90 systolic, 70 diastolic, and the heart rate slowed to 150. One and a half hours later an electrocardiogram showed questionable auricular flutter at a rate of about 300 but not a truly regular auricular or ventricular rate.

There was still a bigeminy of QRS complexes at about 70, with marked variation in conduction and especially aberrance of conduction in the second of the coupled beats.

Two hours after the administration of Cedilanid, 1.2 gm. of quinidine was administered over a period of four hours. An electrocardiogram taken three hours after the beginning of the course of quinidine showed P waves at a regular rate of 90, without relation to QRS complexes, which were wide and slurred, at a rate of 90 to 95. Ventricular premature beats were frequent, and there were occasional groups of them, all from different foci.

Eight hours after admission the blood pressure could not be obtained. The apical rate was regular at 160. An electrocardiogram showed paroxysmal ventricular tachycardia at a rate of 210. The patient expired three hours later, eleven hours after admission.

Differential Diagnosis

Dr. Howard B. Sprague: With only the information given us that could be obtained from the patient, we can hardly do more than make a reasonable catalogue of probabilities, it seems to me, in discussing the background of the one outstanding feature here, namely, the extremely abnormal ventricular rhythm, which started with auricular flutter with an intermittent aberrance of the ventricular complexes and finally passed into a true ventricular paroxysmal tachycardia, or what one may call ventricular flutter, before death. Outstandingly ventricular paroxysmal tachycardia and this terminal rhythm, a ventricular flutter that passed over into true ventricular fibrillation, are found in relation to coronary-artery disease, with myocardial infarction, in digitalis intoxication and at times from digitalis plus quinidine and perhaps from quinidine alone. Obviously with a woman only twenty-nine years of age the first etiology seems to be highly unlikely, nor do we have evidence of toxic doses of digitalis or quinidine. The history starts, not particularly acutely, with attacks of weakness and dizziness of five weeks' duration. They were accompanied by vertigo, nausea and vomiting, which seem to me to be consistent with episodes of abnormal cardiac rhythm and cerebral anemia or perhaps with small embolic or thrombotic affairs in the cerebral circulation. Recently there had been some blurring of vision and probably a diplopia, but this could not be demonstrated as having an anatomic basis at the time of the examination. Simultaneously there was a questionable slight stiffness of the neck. All these findings can be explained by the mechanism I have mentioned. It also appears likely that the abnormal rhythm was at first and fundamentally an auricular tachycardia.

In analyzing the electrocardiogram, auricular waves appear to be present and there is an auricular flutter with probably aberrant ventricular responses which increase in frequency to the point where they take off with an independent rhythm. There is evidence of a re-entry phenomenon, so that true ventricular paroxysmal tachycardia eventually ensues from the re-entrance of the abnormal beats.

There were on physical examination a big heart, a high diaphragm and an enlarged liver, but the neck veins were not engorged and there was slight fever, and some leukocytosis.

Let us return to the catalogue of probabilities — the conditions in which ventricular paroxysmal tachycardia occurs. I have mentioned coronary disease and digitalis intoxication. A few years ago, when heavy doses of digitalis were given, with rapid digitalization, this type of thing did occasionally occur, with fatal results. There is no history that this patient was receiving excessive medication before entering the hospital. Some patients do not know when they are receiving digitalis. I remember a patient with severe digitalis intoxication who denied the possibility of taking the drug. She said that she was taking "only a capsule of white powder." We examined one of these and found a digitalis pill hidden within. She had become toxic without knowing that she was taking digitalis. Quinidine may be a factor in producing this rhythm, although most of the evidence indicates that the way to treat it is by quinidine. About twenty years ago Davis and I[1] reported a case in which we believed that quinidine and digitalis were responsible, in the presence of bundle-branch block, for producing re-entry and fatal ventricular tachycardia. Hypertension is found as a cause, rarely rheumatic heart disease, but not infrequently the examiner finds no evidence of valvular disease in the case, since with these rhythms it is difficult to be sure of the auscultatory findings. Rarely thyrotoxicosis may produce it. It has been described in congenital heart disease with ventricular septal defects, and in the South American literature at least, it is described with gumma of the ventricular septum. Indeed, emphasis on pathology in the septum is rather prominent in autopsy reports of deaths associated with ventricular tachycardia.

One always mentions periarteritis nodosa in these pathological conferences, but I find no evidence for it, although it can do as bizarre things to the heart as does a tumor of the myocardium. An undiagnosable type of interstitial myocarditis or myocarditis of the Fiedler type might be possible, or abscess of the septum of the heart.

There have been, strange to say, cases described in young girls with nothing wrong but obesity. In one case an attempt at a weight reduction program was followed by ventricular paroxysmal tachycardia and death. In another case autopsy showed nothing but a minor fibrosis in the heart muscle. Intoxications from various drugs, chiefly the chlorinated hydrocarbons (carbon tetrachloride, ethyl chloride, tetrachlorethylene, chloroform and cyclopropane),

may produce this type of rhythm. There is also the patient who has ventricular paroxysmal tachycardia in an apparently normal heart. At least those that die may show at autopsy a mild fibrosis of the myocardium. It has been described in cases of calcified pericardium.

Finally there are the diagnoses that one of my naval medical friends calls "those in the back of the book" which can be listed, but I find no leads for any of those diagnoses.

There is some evidence of infection, such as the fever, leukocytosis, tender fornices on vaginal examination, tender liver and high diaphragm. Isolated myocarditis, the Fiedler type, has been described following various infections, including gonorrheal infection. We have no evidence of that here, and no vaginal discharge. There may be some undiagnosable infection of the abdominal cavity, but I do not know where it is.

In conclusion I am going to say that all the ordinary probabilities seem to be absent in this particular case. The evidence is somewhat more in favor of infection of the myocardium of an unknown type. There may perhaps be a lesion of the septum. There should be an enlarged and dilated heart, perhaps more so on the right than on the left.

Was the head examined at autopsy?

DR. MALLORY: No.

DR. SPRAGUE: Small cerebral thrombi may have been present. The patient really died of shock.

I should be interested to know whether the clinicians did any better than I have in deciding on a diagnosis.

DR. WADE VOLWILER: The clinicians did not do so well. The patient apparently had some cardiac disorder, but no murmurs were heard. She was sent into the hospital by her physician as a brain-tumor suspect because of the dizzy spells. On the way to the hospital he discovered the arrhythmia and started digitalis. The ventricular tachycardia appeared when we were giving large amounts of quinidine, and the question came up whether one should stop or give more quinidine. The opinion was that we should not give more since the arrhythmia was produced during quinidine administration.

DR. SPRAGUE: The theory behind quinidine and digitalis poisoning, especially that due to quinidine, is that, in the presence of bundle-branch block, the conducting mechanism of the heart, which permits the ventricles to discharge synchronously and all over at once, is interfered with. This allows parts of the ventricle to recover from their refractory periods at different times, so that where an abnormal beat is propagated through the ventricle it may re-enter through an area in the muscle that has barely recovered, which in turn sets up a circus movement through the ventricle. It may be initiated by the use of digitalis or quinidine or the combination.

DR. ROBERT S. PALMER: I should like to ask Dr. Sprague if he thinks that a large heart like this and tachycardia could occur in a twenty-nine-year-old woman in five weeks without some previous damage in the heart. Perhaps congenital sclerosis of the coronary vessels would produce it.

DR. SPRAGUE: We have no evidence that anything was wrong with the heart previously. The enlargement may have been almost entirely due to dilatation.

CLINICAL DIAGNOSES

Cardiac arrhythmia.
Auricular flutter changing to ventricular tachycardia.
Cerebral embolus?

DR. SPRAGUE'S DIAGNOSES

Acute myocarditis, with involvement of interventricular septum, of unknown etiology (? Fiedler type).
(Auricular flutter.)
(Ventricular tachycardia and flutter.)

ANATOMICAL DIAGNOSES

Idiopathic acute myocarditis (? influenzal myocarditis).
Pulmonary emboli, multiple.
Mural thrombi: right auricle and right ventricle.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The autopsy on this patient showed a heart that was not hypertrophied and that was only moderately dilated. The liver was normal in size. The spleen was almost completely atrophic, weighing only 20 gm. The liver on section showed prominent central markings of the so-called "nutmeg" type. On opening the pulmonary artery, emboli were found in considerable number, and on going back to the heart, we found mural thrombi in the right auricle and right ventricle. The myocardium showed discoloration in numerous areas, particularly, as was prophesied, in the interventricular septum. This showed patchy gray spots that felt rather firmer than normal. Since the coronary arteries were completely normal, we strongly suspected that myocarditis was present. This was later confirmed microscopically.

This is a section of the myocardium (Fig. 1). There are large areas from which the muscle cells have disappeared almost completely and in which only a few swollen hyalinized portions of fibers persist. Over to the right the fibers are still present but are obviously abnormal. The striations have been lost, there is central vacuolization in some, and they stain more intensely than normal. A rather striking feature of the section, which does not show in the photomicrograph because of inadequate magnification, is that some of these persistent muscle fibers contain a great many nuclei, sometimes five or ten in a single cell, much like the multinucleated giant cells one sees in degenerating skeletal muscle.

Mitotic figures are never found in muscle fibers after infancy, but the nuclei multiply amitotically by budding and fission. This occasionally occurs in heart muscle, but by no means so frequently as in striated voluntary muscle.

So we have here a severe myocarditis unassociated with proved infection anywhere else in the body. The valves were entirely negative. There was nothing in any of the organs to suggest rheumatic fever.

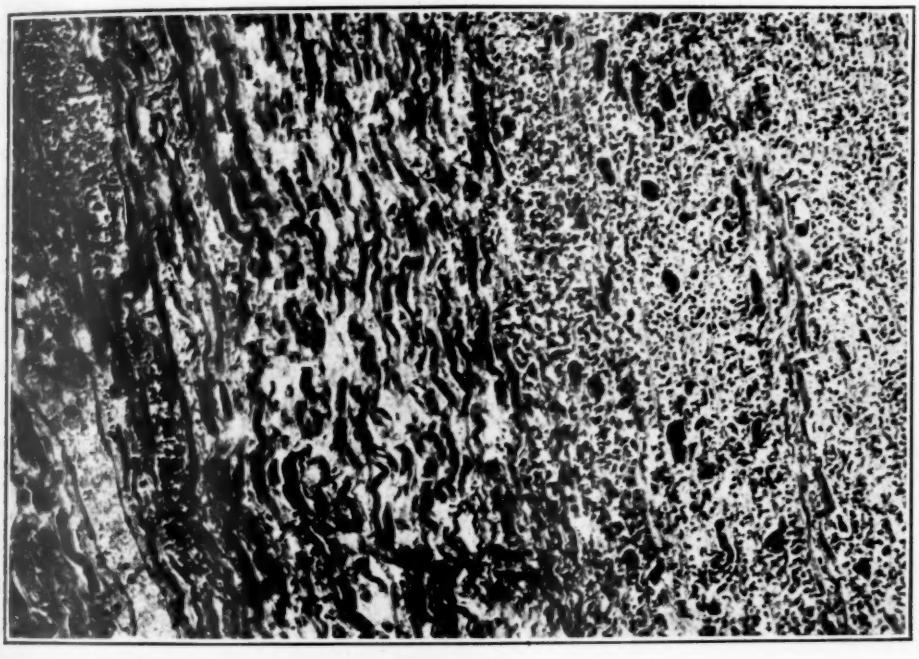

FIGURE 1.

It could be described and catalogued as "isolated," meaning merely that no other pathologic lesion was found in the body to account for it. An equally good term is "idiopathic," because, after all, we know nothing whatever of the etiology in a great many of these cases. Myocarditis does appear in a rather wide variety of infections, such as meningococcal and streptococcal infections, but only irregularly and infrequently. The only common causes of myocarditis are diphtheria and rheumatic fever. It is rare that rheumatic fever produces massive necrosis of muscle such as you see here. Diphtheria commonly does, but there is nothing in the history to permit us to suppose that this girl had diphtheria.

A form of myocarditis due to sulfonamide therapy has become well known to pathologists in the last three or four years. It has not attracted a great deal of clinical attention so far as I am aware. Myocarditis with extensive necrosis of muscle fibers has been attributed to sulfonamide injury.[2] I have never personally seen such a case out of several score of cases of sulfonamide myocarditis. In my experience they have uniformly shown an interstitial myocarditis with little or no degeneration of the muscle fibers. I consider that a highly improbable etiology here.

One other cause of myocarditis was brought to our attention here in Boston a couple of years ago by Finland, Parker, Barnes and Jolliffe,[3] who reported two cases of myocarditis immediately following infection with influenza A virus. Inasmuch as at the present time there is a mild epidemic of influenza A, it is a possibility in this case, perhaps the best guess one can make, but we have no proof of it.

DR. SPRAGUE: As far back as 1921 Fiedler's myocarditis was described following influenza, so that it seems quite likely.

DR. GEORGE C. COTZIAS: If it were trichinosis, would one have any difficulty in finding the organism?

DR. MALLORY: One should have no difficulty in demonstrating the parasites in a case of this severity.

REFERENCES

1. Davis, D., and Sprague, H. B. Ventricular fibrillation: its relation to heart block. *Am. Heart J.* **4**:559-572, 1929.
2. Lederer, M., and Rosenblatt, P. Death during sulfathiazole therapy: pathologic and clinical observations on four cases with autopsies. *J. A. M. A.* **119**:8-18, 1942.
3. Finland, M., Parker, F., Jr., Barnes, M. W., and Jolliffe, L. S. Acute myocarditis in influenza A infections: two cases of nonbacterial myocarditis, with isolation of virus from lungs. *Am. J. M. Sc.* **209**:455-468, 1945.

The New England Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

Richard M. Smith, M.D., *Chairman*
James P. O'Hare, M.D. — Conrad Wesselhoeft, M.D.
Oliver Cope, M.D. — John Fallon, M.D.

Official Organ of
The Massachusetts Medical Society
and
The New Hampshire Medical Society

Editorial Board
Joseph Garland, M.D. — A. Warren Stearns, M.D.
Shields Warren, M.D. — Chester S. Keefer, M.D.
C. Guy Lane, M.D. — Fletcher H. Colby, M.D.
William A. Rogers, M.D. — Robert L. Goodale, M.D.
Henry R. Viets, M.D. — Chester M. Jones, M.D.
Robert M. Green, M.D. — Harvey R. Morrison, M.D.
Charles C. Lund, M.D. — Maxwell Finland, M.D.

Associate Editors
Thomas H. Lanman, M.D. — Donald Munro, M.D.
Henry Jackson, Jr., M.D.
Walter P. Bowers, M.D., Editor Emeritus
Robert N. Nye, M.D., Managing Editor
Clara D. Davies, Assistant Editor

Subscription Terms: $6.00 per year in advance, postage paid, for the United States (medical students, $3.50 per year); Canada, $7.04 per year (Boston funds); $8.52 per year for all foreign countries belonging to the Postal Union.

Material should be received not later than noon on Thursday, two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

NEW A.M.A. PROGRAM

The new National Health Program of the American Medical Association and its scheme to establish minimum standards of acceptance for prepayment medical-care plans and to create a voluntary federation — Associated Medical Care Plans, Incorporated — are described in an editorial in the February 23 issue of the *Journal of the American Medical Association*, which should be thoughtfully read by all physicians.

The National Health Program is a great improvement over the original fourteen-point program approved by the Board of Trustees last summer. Many of the obvious omissions in the latter, some of which were pointed out in an editorial appearing in the August 16 issue of the *Journal*, have been included in the new program. Unfortunately, however, no reference is made to the need for improving the quality of medical care; in other words, it still seems to be taken for granted that all licensed physicians are equally well qualified to care for the sick.

Undoubtedly the standardization of medical-care plans will do much to ensure the success of such organizations, which in the past have enjoyed varying fortunes owing to a lack of sound actuarial data. Such figures are now becoming available, — for example, those concerning Massachusetts Medical Service, a pre-eminently successful venture, — and will be supplied by a new department of the American Medical Association — the Division of Prepayment Medical Care Plans — for the purpose of reorganizing nonacceptable plans or of creating new ones. One of the most significant steps is the formation of the voluntary federation, the chief aim being co-ordination of and reciprocity among all such medical-care plans. This should permit transference of subscribers from one plan to another and the rendering of service to subscribers outside their home states, all of which should encourage the acceptance of such plans by large national industrial organizations. Furthermore, a federation of this sort should be able to conduct a well co-ordinated and effective campaign of public education.

The American Medical Association appears to have initiated a constructive and promising program for improving the health of the Nation and for providing means of meeting the costs of medical care. It is to be hoped that this program will answer much of the criticism that has been directed at so-called "organized medicine" during the last few years.

"TO DO SOMETHING FOR THE WELFARE OF MANKIND"

The twenty-seventh annual report of the Commonwealth Fund accounts for the spending of $1,418,056. This sum is divided between the Fund's special programs and a group of more diverse and various grants. The largest amounts in the first category were appropriated in the interest of public health and the further extension of the now well known rural-hospital program. Another hospital

to be constructed, in Sikeston, Missouri, and a new experiment in regional medical organization is planned for the area surrounding Rochester, New York. Smaller, but still significant amounts were devoted to mental health, education and publications. The special grants for medical research, war relief and miscellaneous purposes total $677,457. By far the greater amount of the latter expenditures continued to be for medical interests and activities. These are the items that appear at the end of the report, where they are often read first by those interested in where and how money is being awarded. It is probably intended, however, that a reading of the foreword precede indulgence in the gross figures of distribution.

As in previous reports, the foreword suggests why money is being spent where it is. It is a thumbnail essay on the difficulties and uncertainties that currently face medical practice and education. It contains several incisive observations, such as, "No short-cut to the integration of specialized knowledge into comprehensive medical care has yet been devised . . ." and ". . . the practical details for a coherent plan for the encouragement and support of research remain to be worked out." It is then recognized that these are serious questions and that they are not likely to be solved in financial terms alone. Although the foreword emphasizes that what the Fund can do is a small part of what must be done, it states the minimum objectives of the Fund as follows: sound training for professional and technical workers in strategic phases of health maintenance; the broadening and integration of the basic concepts of medical care so that it may become truly comprehensive; and the practical encouragement of human curiosity as the fountainhead from which medical knowledge renews itself.

The health of the Nation is a vast borderland between a philosophy and a discipline, between sociology and medicine. In it there are many mansions, some old ones with outworn and useless concepts, some new ones, unfinished and with untried architecture. There are other comfortable and livable houses in which the tenants have kept in touch with the practical realities of the world and are contributing to the evolution of the human race. The Commonwealth Fund continues to house such tenants.

MASSACHUSETTS MEDICAL SOCIETY POSTWAR LOAN FUND

The Postwar Loan Fund has been set up, and all discharged medical officers who were members of the Massachusetts Medical Society in good standing at the time of their entry into the service may apply for loans from this fund. For further information apply to:

George L. Schadt, *Chairman*
Postwar Loan Fund
8 Fenway
Boston 15, Massachusetts

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

ALEXANDROV — Vitaly J. Alexandrov, M.D., of Rutland, died March 5. He was in his fifty-sixth year.

Dr. Alexandrov received his degree from Tufts College Medical School in 1930. He was born in Russia and graduated from the University of Moscow. He was consultant and medical director of the tuberculosis unit of Burbank Hospital, Fitchburg, director of the Fitchburg Tuberculosis Clinic, and physician-in-charge of the Rutland Cottage Sanitarium. He was director of the Northern Worcester County Public Health Association. He was a fellow of the American College of Chest Physicians, the American Trudeau Society and the American Medical Association.

His widow, two daughters and two sons survive.

BURGESS — Charles J. Burgess, M.D., of Lawrence, died February 16. He was in his seventy-seventh year.

Dr. Burgess received his degree from Tufts College Medical School in 1898. He was a member of the Roentgen Ray Society and the Radiological Society of North America, Incorporated, and a fellow of the American Medical Association.

His widow survives.

SCENNA — Donato T. Scenna, M.D., of Melrose, died March 3. He was in his forty-second year.

Dr. Scenna received his degree from Tufts College Medical School in 1930.

His widow survives.

MEDICOLEGAL ABSTRACT

Regulation of Practice by Government: Power of government. The plaintiff was incorporated as a nonprofit corporation. The corporation had three classes of members: administrators, practicing physicians and beneficiary members, who on pay-

ment of monthly dues, were entitled to the professional services of the participating physicians. It was provided that all compensation for doctors would be on a pro rata basis out of monthly dues. The corporation had about 5000 members in the second class and about 100,000 members in the third class; the latter was growing at a rate of 1500 members a month.

The plaintiff brought an action for a declaratory judgment to determine whether it was engaged in an illegal corporate practice of medicine and whether its activities were in the nature of disability insurance subject to certain sections of the insurance code.

On the first question, the California Supreme Court held that the prohibition against the corporate practice of medicine does not apply to nonprofit organizations. (The same doctrine has recently been expressed by way of implication in Texas where the court said by way of dictum that a corporation organized for profit cannot practice medicine and that a license to practice will issue only to a natural person. — *Woodson* v. *Scott and White Hospital*, 186 S.W. [2d] 720, 1945, Texas.

On the second issue, the California court went into considerable detail in finding that the plan set forth did not constitute insurance. It found the group medical plan analogous to a consumer co-operative, saying in part:

> Neither as between the corporation and the physician, nor as between the physician and the subscriber, is the compensation or any other element of the arrangement between them affected by any contingency, hazard or risk.
>
> The latter [insurance companies] are concerned primarily, if not exclusively, with risk and the consequences of its descent, not with service or its extension in kind, quantity or distribution; with the unusual occurrence, not the daily routine of living. Hazard is predominant. On the other hand, the co-operative is concerned principally with getting service rendered to its members and doing so at lower prices made possible by quantity purchasing and economies in operation. Its primary purpose is to reduce cost rather than the risk of medical care; to broaden service to the individual in kind and quantity; to enlarge the number receiving it * * * not except incidentally * * * to indemnify for cost after the service is rendered.

In 1941 the plaintiff entered into a contract with the Federal Farm Security Administration to furnish medical services and hospital care to rural families. The state insurance commissioner attempted to show that the contract to provide hospitalization was insurance. For procedural reasons this question was not decided. The court did, however, say that a single aspect of a plan could not be singled out for condemnation, that the plan must be looked at as a whole, even though a contract to provide hospitalization, when viewed alone, would under some circumstances be insurance. — (*California Physicians Service* v. *Garrison*, 155 P. [2d] 885, Feb. 15, 1945; rehearing denied Mar. 17, 1945; hearing granted Apr. 12, 1945.)

NOTICES

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, April 4, at 7:15 p.m. in the classroom of the Nurses' Residence. Dr. Bertha Offenbach will speak on the subject "The Common Eye Diseases Among Children."

TUFTS MEDICAL ALUMNI ASSOCIATION

The annual dinner meeting of the Tufts Medical Alumni Association will be held on Wednesday, April 10, at the Somerset Hotel, Boston. The committee on arrangements consists of Drs. George W. Holmes, Alton S. Pope, Samuel A. Robins, A. Warren Stearns, John J. Todd, Edwin T. Wyman and Harry Blotner.

The guest speaker will be Major General Norman T. Kirk, who will speak on the subject "American Medicine in World War II." A message will be read from President Harry S. Truman. Other speakers will include Dr. Reginald Fitz, president of the Massachusetts Medical Society, Dr. A. Warren Stearns and Dr. Dwight O'Hara, Dr. A. K. Paine, professor of obstetrics, Dr. Louis E. Phaneuf, professor of gynecology, Dr. Benjamin Sachs, professor of ophthalmology, and Dr. Leonard Carmichael, president of Tufts College. Dr. William E. Browne, professor of clinical surgery, will preside.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, MARCH 28

FRIDAY, MARCH 29
*9:00–10:00 a.m. Some Aspects of Military Orthopedics Applicable to Civilian Practice. Dr. Joseph S. Barr. Joseph H. Pratt Diagnostic Hospital.
*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.
10:50 a.m. Virus Diseases of the Skin. Dr. George E. Morris. (Postgraduate Clinic in Dermatology and Syphilology.) Amphitheater, Dowling Building, Boston City Hospital.

MONDAY, APRIL 1
*12:00 m.–1:00 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

TUESDAY, APRIL 2
*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

WEDNESDAY, APRIL 3
*10:30–11:30 a.m. Medical Clinic. Isolation Building Amphitheater, Children's Hospital.
*12:00 m. Clinicopathological Conference (Children's Hospital). Amphitheater, Peter Bent Brigham Hospital.
*2:30–4:00 p.m. Combined Clinic by the Medical, Surgical and Orthopedic Services. Amphitheater, Children's Hospital.

*Open to the medical profession.

JANUARY 7–APRIL 22, 1946. Metropolitan State Hospital. Eleventh postgraduate seminar in neurology and psychiatry. Page 314, issue of September 6.

FEBRUARY 4–MARCH 29. Health Education Institute. Page 746, issue of December 13.

MARCH 15–SEPTEMBER 15. Boston University Course for Discharged Medical Officers. Page 240, issue of February 14.

MARCH 25–27. Medical Library Association. Page 356, issue of March 7.

APRIL 1–JUNE 1. Intensive Course in Ophthalmology. Page 240, issue of February 14.

APRIL 4. Hermann M. Biggs Memorial Lecture. Page 206, issue of February 7.

APRIL 4. New England Hospital for Women and Children. Notice above.

APRIL 10. Tufts Medical Alumni Association. Notice above.

APRIL 11. Breast Tumors: Problems of diagnosis and treatment. Dr. Joseph Tartakoff. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

MAY 6–11. American Board of Obstetrics and Gynecology. General oral and pathological examinations. Palmer House, Chicago.

MAY 13–17. American College of Physicians. Page 798, issue of December 20.

JUNE 20–22. American Association for the Study of Goiter. Page 312, issue of February 28.

DISTRICT MEDICAL SOCIETY

WORCESTER

APRIL 10. Hahnemann Hospital.
MAY 8. Annual meeting.